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THE EXCRETION OF INTRAVENOUSLY INJECTED SODIUM THIOSULPHATE DURING UNCOMPLICATED HUMAN PREGNANCY.

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INTRODUCTION.

NINE years ago Nyiri⁽¹⁾ introduced the use of sodium thiosulphate as a means of estimating the renal function. In brief, the principle of the test, which found a good deal of favour, is as follows.

In health, after the intravenous injection of sodium thiosulphate, a certain proportion is excreted unchanged by the kidneys, the remainder most probably being oxidized in the body to sodium sulphate.⁽²⁾ In the presence of an impaired renal function the percentage excretion becomes impaired to an extent varying with the severity of the lesion. During an experimental study of the test by one of us,⁽³⁾ it was observed, so far as we can ascertain for the first time, that in the pregnant animal there also occurred an impairment of excretion, to account for which no discoverable element of renal dysfunction was present. A further and more detailed study of this aspect of the test was subsequently carried out in the pregnant bitch⁽⁴⁾ and there was found a constant depression of the thiosulphate excretion, commencing about three weeks after conception and lasting till term, after which in uncomplicated

pregnancy it quickly returned to normal. In view of these findings it was decided to carry out a series of estimations on the human subject, with the object of discovering whether a similar depression would be encountered, and, if so, to ascertain its extent, significance and possible clinical application.

TECHNIQUE.

Type of Patients Studied.

The investigation was carried out on a series of pregnant patients, suffering from venereal disease, admitted to the Royal Prince Alfred Hospital some time before the expected date of confinement. Both before and after confinement estimations of the thiosulphate excretion were carried out as regularly as was practicable. A series of non-pregnant patients from the same ward was also studied for the purpose of establishing controls. All of these patients were under treatment according to the indications present.

Preparation of Thiosulphate Solution.

As in previous work more than one dosage was employed. In order to obtain quickly a clear and sterile solution of sodium thiosulphate the following procedure was adopted.

One hundred cubic centimetres of sterile distilled water were boiled for a few minutes in a sterile flask. The heating was then stopped and either ten, twenty or thirty grammes of crystalline sodium thiosulphate B.P. ($\text{Na}_2\text{S}_2\text{O}_3 \cdot 5\text{H}_2\text{O}$) were added, and boiling was resumed for a few more minutes. The bottle was then stoppered with a piece of sterile cotton wool. Cloudiness of the solution occasionally necessitated filtration, after which it was once more brought to the boil. After cooling, the solution was ready for injection. A fresh solution was always prepared immediately before each batch of injections.

On account of the increase in volume due to the addition of the salt, ten cubic centimetres of the above solutions respectively contain approximately 0.95 gramme, 1.8 grammes and 2.6 grammes of crystalline sodium thiosulphate. The last mentioned, of which most use was made, represents a solution of about twice normal strength.

Procedure and Chemical Technique.

Ten cubic centimetres of one of the solutions of sodium thiosulphate prepared as described were injected intravenously on the occasion of each test. The injection must be entirely intravenous, since, besides vitiating the results, any escape of solution into the subcutaneous tissues was found to cause a good deal of pain, not accompanied, however, by any subsequent objective signs. After a proper intravenous injection no ill effects were ever observed.

Each patient was given about two hundred cubic centimetres of water at the time of injection, one and two hours after which specimens of urine were collected for thiosulphate estimation. Catheterization was not carried out, the patients merely being instructed to empty the bladder completely.

For the purpose of estimating the sodium thiosulphate output, approximately a teaspoonful of

Fuller's earth, or preferably Lloyd's reagent (a proprietary preparation of purified and concentrated Fuller's earth), was added to the urine, diluted with water to a convenient volume, usually five hundred cubic centimetres. The mixture was then shaken and filtered, and a known fraction of the filtrate (ten to fifty cubic centimetres) acidified with normal sulphuric acid and titrated against a one-hundredth normal iodine solution, starch being used as the indicator. After each series of injections ten cubic centimetres of the thiosulphate solution left over were similarly diluted and titrated.

The number of cubic centimetres of one-hundredth normal iodine solution decolorized by the thiosulphate content of the urine, divided by the number decolorized by the amount of thiosulphate injected and multiplied by 100, gives the percentage of the injected thiosulphate which has been excreted. That is:

$$X = \frac{\text{C.cm. N/100 I required by urine} \times 100}{\text{C.cm. N/100 I required by } \text{Na}_2\text{S}_2\text{O}_3 \text{ injected.}}$$

On account of the iodine-combining substances normally present in the urine, the figures so obtained do not quite represent the true percentage excretion of sodium thiosulphate, and minor corrections should preferably be made by small deductions. Assuming that the iodine-combining substances normally present remain stationary, their significance will naturally diminish in proportion as the dosage of sodium thiosulphate is increased. We made deductions from our own figures for the two-hours' excretion as follows:

$$\begin{aligned} \text{After 0.95 gramme} &= 2.2\% \\ \text{" 1.8 grammes} &= 1.2\% \\ \text{" 2.6 grammes} &= 0.8\% \end{aligned}$$

These figures represent the mean value obtained by titrating a series of normal two-hour excretions of urine against a one-hundredth normal iodine solution, expressed as percentages of the values obtained by similarly titrating the standard thiosulphate solutions. The total two-hours' percentage excretion, arrived at by the above means, is expressed throughout this paper as the nearest whole number.

EXPERIMENTAL FINDINGS.

The Excretion of Sodium Thiosulphate in Non-Pregnant Patients.

It has been previously observed by one of us that in dogs⁽⁴⁾ the amount of thiosulphate injected has some influence on the percentage excreted. This was also observed by Holbøll⁽⁵⁾ in the human subject after the administration to five normal individuals of amounts of thiosulphate representing from 0.78 gramme to 6.28 grammes of the crystalline salt. With other workers the dosages used varied little. Table I summarizes the results obtained by these various workers and by ourselves. To facilitate comparison with our own figures, their dosages are uniformly expressed as grammes of crystalline sodium thiosulphate ($\text{Na}_2\text{S}_2\text{O}_3 \cdot 5\text{H}_2\text{O}$).

In a large series of one hundred and sixty-two normal and non-renal subjects, Nyiri⁽¹⁾, after the injection of 1.57 grammes, found a two-hour excretion of sodium thiosulphate ranging from 20% to 43%.

TABLE I.

Summary of Results obtained up to the present by Different Authors following Intravenous Injection of Sodium Thiosulphate.

Author.	Number of Cases.	Grammes of $\text{Na}_2\text{S}_2\text{O}_3 \cdot 5\text{H}_2\text{O}$ Injected.	Range of 2-hour Excretion (%)	Mean 2-hour Excretion (%)
Nyiri ⁽¹⁾	162	1.57	20-43	
Iverson and Fasting-Hansen ⁽⁶⁾	24	1.57	20-41	
Eskelund ⁽⁷⁾	10	1.57	23.3-34.8	
Humbert and Finck ⁽⁸⁾ . . .	?	1.24	23.4-30.7	25.9
Silberstein ⁽⁹⁾	?	1.24		29.9
Holbell ⁽⁵⁾	5	0.78	15.0-21.3	18.9
	5	1.57	25.4-29.8	27.6
	3	3.14	33.4-46.3	37.5
	4	4.71	40.4-57.3	45.3
	5	6.28	48.7-60.8	57.7
Bolliger and Earlam . . .	14	0.95	19.0-31.2	23.7
	13	1.82	25.0-40.5	30.6
	12	2.58	25.1-45.9	31.9

The findings in five communications from other workers (Iverson and Fasting-Hansen,⁽⁶⁾ Eskelund,⁽⁷⁾ Humbert and Finck,⁽⁸⁾ Silberstein⁽⁹⁾ and Holbell⁽⁵⁾) who injected either the same amount or somewhat less, namely, 1.24 grammes, fall within the same limits. Authors dealing only with healthy individuals (Humbert and Finck, Silberstein, Eskelund and Holbell) obtained a considerably narrower range of values. Holbell demonstrated on his small series of normal individuals that the percentage of thiosulphate excreted increases with increasing amounts injected. Our group of patients also demonstrates this principle. It will also be observed that although the amounts injected by us differ from those used by other workers, our findings are within limits in accord with those obtained by them on normal and non-renal cases. Similarly we found also that the bulk of the excretion always took place during the first hour.

In order to evaluate our subsequent findings in the presence of pregnancy, we laid down, in accordance with the observations made by ourselves and others, the following figures as the lowest normal values:

After injection of 0.95 gramme, lowest normal value = 19%
 " " 1.8 grammes, " " = 24%
 " " 2.6 grammes, " " = 26%

At this stage it should be mentioned that out of forty-six non-pregnant patients suffering from venereal disease who were examined, seven showed an excretion which was below the established normal, some even showing less than half of the minimum normal excretion. These patients were all suffering from various complications of their original venereal infection, and do not fall within the scope of this paper. They will be discussed elsewhere.

The Excretion of Sodium Thiosulphate in Uncomplicated Pregnancy.

We examined the thiosulphate excretion in thirty-seven pregnant patients both before and after delivery of a full-term child. In nine of these we were able to make only one determination, and two determinations only in five. But in nine cases observations were made on from six to thirteen occasions. These cases necessarily supply the body of our observations, while those where only a few determinations were made amplify the points of interest. According to the results we obtained, we subdivided our cases into the following two groups:

- (I) Pregnancies with no or only short-lived depression in the thiosulphate excretion.
- (II) Pregnancies with marked and prolonged depression in the thiosulphate excretion.

(I) Pregnancies with No or only Short-lived Depression in the Thiosulphate Excretion.

Table II represents the sixteen cases in which at least two estimations were made, and which we classed into this group. The findings have been arranged according to the *ante partum* and *post partum* weeks in which they were obtained. The child in every case was full-term, and for the sake of simplicity labour has been listed in the table as occurring at the thirty-ninth week. An asterisk (*) opposite any figure denotes an injection of 0.95 gramme, a circle (°) one of 1.8 grammes, while all other figures were obtained after the injection of 2.6 grammes.

The findings before the twenty-third week are not incorporated in the table, and will be briefly mentioned. In Case XIV six determinations were made between the twelfth and twenty-third weeks of pregnancy. The injections were of 2.6 grammes and resulted in the excretion, in chronological order, of 38%, 34%, 39%, 36%, 24% and 23%, the last two figures being slightly subnormal. In Case IX, 27% was excreted in the twenty-first week, following the injection of 0.95 gramme. In three other cases, not appearing in Table II, high normal values were obtained between the tenth and twenty-first weeks.

But of the sixty-two tests listed in Table II, sixteen showed subnormal values in the *post partum*, as well as in the *ante partum* period. None of these subnormal values were found to occur from the twenty-third to the thirty-first week. But from the thirty-second to the thirty-fourth week, in eight cases five instances of definitely subnormal values were found. For example, Cases X and XIII showed a depression of 10% below the minimum normal value in the thirty-fourth and thirty-third weeks respectively. In three cases (III, VII and VIII) in which determinations were made during this period there were no subnormal values; in two of these only one estimation was carried out. During the next three weeks there is a tendency towards normal excretion. Depressed values are met with (Cases II, IV, XI and XV), but the depressions are distinctly less outspoken than those previously occurring.

In five instances determinations were made less than a week before confinement. These results are listed between the thirty-eighth week and confinement,

TABLE II.
Excretions of Sodium Thiosulphate during Pregnancy and the Puerperium in Patients of Group I.

Case.	No.	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	1	2	3	4			
K.M.	1	27*	37*	23*		34*																			
W.H.	2														16*					19*					
H.H.	3												25*				24*	19* ¹							
G.E.	4															18*				29*					
W.D.	5														37*								15*		
H.F.	6								23*		10*		28*							25*	15*			22* (4/12)	
K.K.	7	29*												34°	30°	27°	30°	30°							
W.M.	8							27°		31°	35	28	32	27		31		22*							
S.H.	9							34°	34°	30															
P.S.	10						34°							16	29		25		28 ²						
B.A.	11													21		35	24	20	32 ²				35		
P.V.	12															33								30	
R.J.	13									30	16	30			30					31					
M.A.	14													24	26										
C.A.	15												19		29			22	30						
K.B.	16																	33	36 ²				14	33	

TABLE III.
Excretions of Sodium Thiosulphate during Pregnancy and the Puerperium in Patients of Group II.

Case.	No.	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	1	2	3	4			
E.O'N.	17								20*				14*						11*						
N.N.	18														12*		11*				22*				
I.A.	19						23*							15°		12*			9**	12**	8*	7*		13(14/12) 27(14/12)	
B.F.	20								5*	12*				6*	8*	11°		12°	9°	16° ²	19° ²	24°	26°	14°	27°
S.L.	21												14°	25°	13°	22°	22°	16°	18°		18**				
B.V.	22										21		20	25	21	12	17	16		16			9	8	8 (4/12)
S.E.	23								34	27	9	7	22	20		14	6	13		8			19	8	12

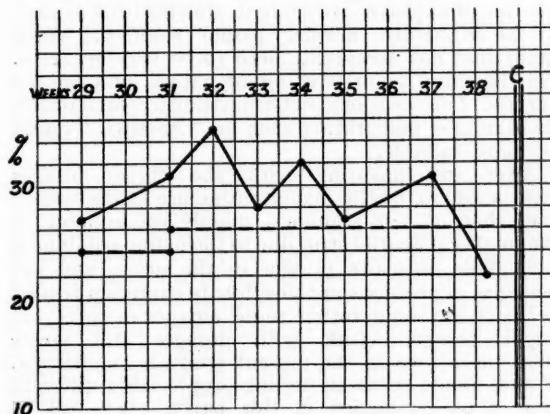
The double vertical line indicates the time of confinement.

the figure accompanying each result indicating the number of days before confinement at which the test was carried out. The figures obtained in this critical period do not show a clear-cut tendency. Cases VIII and X, six and two days respectively before delivery, demonstrate slightly subnormal values, while others (Cases XI and XVI) show somewhat elevated normal results. In Case III a test was performed one day before delivery. Compared with previous findings the thiosulphate excretion was depressed, but still within normal limits.

In this group the number of determinations during the puerperium was small, eleven determinations in eight cases. The tests were performed from one week to four months after confinement, but mostly in the first four weeks. Eight of the findings were within normal limits, the remaining three (Cases V, VI and XVI) very definitely subnormal.

As in the normal patient, the bulk of the thiosulphate excretion in this group occurred in the first hour, during which the excretion in every case quite overshadowed that of the second hour, which ranged from nil to 9%. The great majority of the second hour excretions were between 1% and 2%.

To facilitate comparison of the patients of this group with those of Group II, to be discussed below, the thiosulphate excretion in Case VIII is depicted graphically in Chart I.



W.M., Case VIII. Thiosulphate excretion from twenty-ninth week of pregnancy till just before confinement. Dotted line indicates minimum normal excretion. C=confinement.

This patient, a *primipara* aged nineteen, was suffering from secondary syphilis, for which she was receiving treatment with "Novarsenobenzol" and "Muthanol." Pregnancy and labour were uneventful, and a healthy full-term child was born and survived.

Apart from the presence of venereal disease, all other pregnancies in this group were similarly uneventful.

(II) Pregnancies with Marked and Prolonged Depression in the Thiosulphate Excretion.

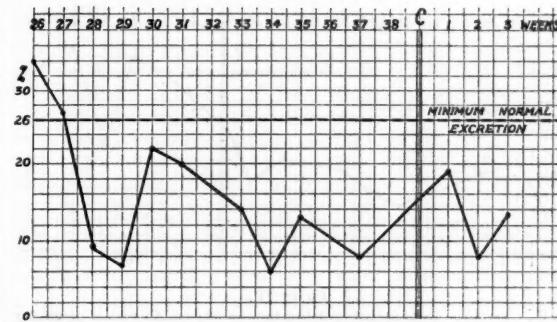
The second group is composed of seven cases, which are listed in Table III, where the findings

are tabulated in a similar manner to those in Table II. In the large majority of these cases fairly numerous estimations were carried out from the twenty-sixth week of pregnancy till various stages after confinement, ranging from one week to fifteen months.

All these patients, with the single exception of the patient in Case XXI, show marked and persistent depression from the twenty-eighth week of pregnancy till confinement. Moreover, in all those patients in whom subsequent estimations were carried out, with one exception again, namely, Case XVIII, the depression persists for varying periods after delivery. Consequently, the percentage of subnormal values in Group II is much higher than in Group I; out of forty-one tests carried out from the twenty-eighth week to delivery, only two gave normal results. Only three tests were carried out before the twenty-eighth week, in Cases XIX and XXIII. These appear in the table, and returned normal values.

As already observed in Group I, some of the patients of Group II tend to have a specially lowered *ante partum* excretion in the thirty-second to thirty-fourth week period (Cases XX, XXII and XXIII). In other respects the different cases show appreciable individual variations, and some will be described in detail.

Case XXIII was that of a *primipara*, aged seventeen, suffering from secondary syphilis and gonorrhoea, for which she received treatment from the seventh month onwards, receiving injections of "Novarsenobenzol" and "Muthanol." Pregnancy, labour and the puerperium were uneventful, and her offspring was a healthy male. The behaviour of the thiosulphate excretion is depicted in Chart II. She received injections of 2.6 grammes. From a normal value at the twenty-seventh week of pregnancy the thiosulphate excretion rapidly fell in the course of a week to the low level of 9%, and to 7% a week later. After a temporary recovery, which, however, fell short of normality, a minimum excretion of only 6% was reached at the thirty-fourth week. From this period till the third week after confinement, when the last estimation was made, the excretion fluctuated, but showed some slight tendency towards improvement, but the last test yielded only 13%, exactly half the value of the minimum normal excretion.



S.E., Case XXIII. Thiosulphate excretion from twenty-sixth week of pregnancy till third week after confinement.

The patient in Case XXII, depicted in Chart III, also received injections of 2.6 grammes. Both mother, aged twenty-two, and child had congenital syphilis, the child dying about thirty hours after birth from intracranial injuries received during birth. Pregnancy and puerperium were uneventful. The mother had previously been under antisyphilitic treatment for a period of two years. At the twenty-eighth week of pregnancy the first estimation of the thiosulphate excretion returned a sub-

normal finding of 21%. From this point the excretion fluctuated, with a general downward trend, till four days before confinement it reached the low value of 10%. Four weeks after confinement the value was still only 8%, and even after three months it was still at the same low level.

Case XX, depicted in Chart IV, is that of a girl aged eighteen, who suffered from gonorrhoea. Pregnancy, labour and puerperium were uneventful, and the child was healthy and survived. This patient had injections of 0.95 grammes up till the thirty-fourth week, after which the dosage used was 1.8 grammes. The first estimation, at the twenty-eighth week, revealed a very markedly depressed excretion of only 5%, about one-fourth the minimum normal value. It remained low throughout the remainder of the pregnancy, rising to a maximum value

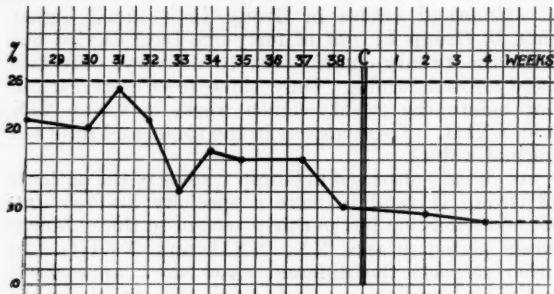


CHART III.

B.V., Case XXII. Thiosulphate excretion from twenty-eighth week of pregnancy till four weeks after confinement. The low excretion seen at this stage was still present three months later. Dotted line indicates minimum normal excretion.

of 16% two days before confinement. Five days after confinement it was higher again, and a week later it reached the minimum normal level of 24%. Another subnormal value of 14% was met with four weeks after confinement, but the excretions at the third and the fifth weeks were normal.

Case XIX, depicted in Chart V, is that of a girl aged seventeen, who was suffering from gonorrhoea. Pregnancy, labour and puerperium were uneventful, and a healthy infant was born which died several weeks after being discharged from hospital. Throughout pregnancy till the fourth week of the puerperium injections of 0.95 grammes were given. Later injections of

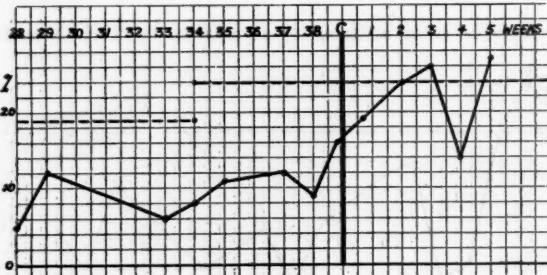


CHART IV.

B.F., Case XX. Thiosulphate excretion from twenty-eighth week of pregnancy till fifth week after confinement. Dotted line indicates minimum normal excretion.

2.6 grammes were employed. At the twenty-sixth week of pregnancy there was a normal excretion of 23%. The next estimation, at the thirty-fourth week, showed a value of 15%, after which a steady fall occurred to 9% two days before delivery. Six days later it had risen slightly to 12%, but again fell gradually to 7% four weeks after confinement, when the patient was discharged from hospital. Twelve months later the excretion was still only 13%, half the normal value, and three months later again a normal excretion of 27% was found, for the first time since the twenty-sixth week of pregnancy.

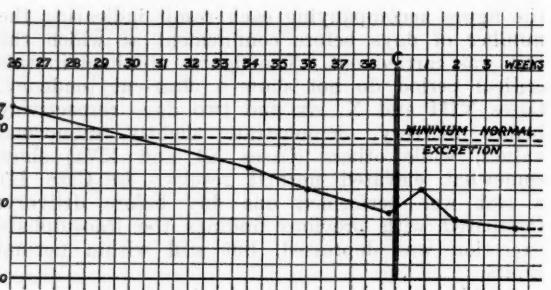


CHART V.
I.A., Case XIX. Thiosulphate excretion from twenty-sixth week of pregnancy till fourth week after confinement. The excretion was still markedly subnormal twelve months later.

In the remaining three cases less determinations were carried out. They demonstrate a practically constant low excretion during pregnancy, and reproduce in part the findings in the cases described in detail. Again, in each of the three cases, pregnancy, labour and puerperium were uneventful, and the child was full-term and healthy.

COMMENT.

Technique.

The instability of thiosulphate solutions led us to the technique described. Rather than use stock solutions which might deteriorate, we considered it preferable to make up from time to time a thiosulphate solution of approximate content, standardized against a one-hundredth normal iodine solution, which again does not necessarily need to be very accurate. This technical point is of minor nature, and for the purpose of an occasional determination a commercially standardized preparation such as "Ametox" would be quite suitable.

After experimenting with the three dosages mentioned, we finally made routine use of the largest one, that of 2.6 grammes, mainly on account of the smaller potential error due to the iodine-combining substances normally present in the urine, and the fact that depressions in thiosulphate excretion tended to be if anything rather more outspoken after the larger dosage. Work with dosages still larger than those used in the present study is in progress. If this should result in findings appreciably different from those embodied in this paper, they will be reported.

Generally speaking, our technique differs only in unimportant details from that employed by other workers, with the exception of our use of a preparation of Fuller's earth in preference to charcoal for the reason described in a previous communication.⁽³⁾ The procedure we have adopted, although very accurate, much more so than for example a colorimetric determination such as the phenolsulphonephthalein test, makes one independent of accurately standardized solutions. As is evident from the results obtained by ourselves and other workers following the use of various dosages, a slight variation in the amount of thiosulphate injected is not sufficient to alter appreciably the percentage excretion.

As with other similar tests, the two principal sources of potential technical error are during the

injection of the solution and in the collection of the specimens of urine. The injections which resulted in the present series of figures were all purely intravenous. In the occasional case where some solution escaped into the subcutaneous tissues, the injection was stopped and the performance of the test deferred. As regards the second source of error, that of incomplete emptying of the bladder, the thiosulphate remaining in the bladder after collection of the first hour's specimen would certainly result in a comparatively high apparent second hour's output, probably of about equal value to that of the first hour. This, however, did not take place, since even with the lowest excretions it was unusual to have a second hour's excretion greater than one-fourth of that of the first hour, and in only one instance did they approximate, in Case VI at the thirty-second week, which estimation alone we therefore regard as possibly not reliable.

Type of Patient Used in the Present Investigation.

At the Royal Prince Alfred Hospital the only group of pregnant patients available for study are those suffering from venereal disease. They are mostly admitted about two months before the expected date of confinement, after which they remain in hospital for another month. To these patients, by the courtesy of the staff, we were given free access.

It may be presumed that the changes observed in the thiosulphate excretion are primarily due in some way to the venereal disease. That this is not the case, however, is suggested by the following facts. A large group of non-pregnant venereal patients completely reduplicating any of the clinical syndromes seen in the pregnant series returned results within normal limits. A second much smaller group did return subnormal values—these were all suffering from complications of their original infection, and, as already mentioned, will be dealt with on another occasion. Furthermore, the only common factor in the present study to account for the depression was the presence of pregnancy. Again, in a group of non-venereal patients being studied elsewhere in connexion with toxæmias of pregnancy, similar depressions have been noted from time to time. Lastly, a series of pregnant bitches previously reported on showed consistent depressions. For these reasons we consider the depressions reported in the present series of cases as being primarily due to pregnancy.

Experimental Results.

We arbitrarily divided our cases of pregnancy into two groups according to the magnitude and duration in the depression of the thiosulphate excretion. The division is not hard and fast as all grades of depression occur from none at all, in Case VII, to prolonged and extreme depression. In Group I, diminished excretions occurred from time to time during the latter months of pregnancy, and transient low values were again met with in the puerperium. But in general, so far as can be made out from the few cases in which estimations were done, the excretion of sodium thiosulphate shows little tendency to persist at a subnormal level after the puerperium.

Cases XVII and XXI of Group II show about the least depression in this group during pregnancy, but they are outspoken enough to justify their inclusion in the group. Similar but more outspoken depressions are found in the other cases. During the puerperium there are distinct differences between the cases of Group II. For instance, while the patient in Case XX, who had shown well-marked and prolonged depression during pregnancy, quickly returned to normal after confinement, the patient in Case XXII showed a definite further impairment of thiosulphate excretion, which reached its lowest level three weeks after confinement, and the patient in Case XIX, after a slight immediate improvement after confinement, also reached a lower level than previously and was still subnormal as long as twelve months later.

The reasons for the depression of the thiosulphate excretion, for the varying degrees of depression among different patients, and for the widely varying periods of time necessary for the complete reversion to normal of the thiosulphate excretion, are alike obscure. In this respect the phenomenon reminds us of the benign glycosuria of pregnancy.

In none of the cases of either of the two groups was there any evidence of renal insufficiency or toxæmia, the pregnancy in every instance being quite uneventful and the venereal disease being well controlled by appropriate treatment. We consider that a temporary depression of the thiosulphate excretion in the last three months of pregnancy is frequently a physiological process with no pathological significance. At this stage we are inclined to regard patients with marked and prolonged depression of the thiosulphate excretion as exhibiting to a more marked degree than occurs in the average case, evidence of the general metabolic disturbances normally associated with pregnancy. These disturbances are, of course, evident in other ways. For example, a subnormal blood urea content is a normal finding during pregnancy, and Gardner and Gainsborough⁽¹⁰⁾ have shown that there occurs a marked disturbance of the cholesterol metabolism. Again it may be quite rational to assume that thiosulphate in view of its recognized detoxicating capabilities may partly be expended in carrying out this function. Some connexion between the cause of the onset of labour and the depression of thiosulphate excretion was looked for, but the results in our series do not support it. In some cases (III, VIII, XIX and XXII) there was a specially marked depression a few days before delivery, but in other instances where tests were done during the week preceding this event the results either remained stationary or showed some improvement as compared with the findings in previous weeks.

With regard to the findings after confinement, it may be pointed out that in Case XIX, nearly a year after lactation had ceased and menstruation had become re-established, there was still a distinctly subnormal output. Also the patient in Case XXII, whose infant died very soon after birth and who resumed menstruation almost immediately, continued to show a definitely subnormal thiosulphate excretion, while the patient in Case XX, who was nursing her baby,

after a marked depression during pregnancy, showed after confinement only some fluctuation similar to that seen in Group I, that is, a single depressed value at the fourth week after a previous return to normal. Again, the patient in Case XXIII, who also nursed her baby, showed as far as examined persistently low values, with a slight tendency for improvement one week after confinement. Again we shall confine ourselves to stating the findings in the hope that subsequent experiments may throw some light on the aetiology of this behaviour of the thiosulphate excretion after confinement.

It may be mentioned here that we were unable to obtain any assistance from the literature in explaining these phenomena. As far as we can determine, only one worker, Clauser,⁽¹¹⁾ has reported on the thiosulphate test during pregnancy. This original article is not obtainable, but it is reported by Stander⁽¹²⁾ that:

He concludes that a definite decrease in renal function, which in normal pregnancy is very slight, becomes marked during labour; and that during the puerperium the kidney usually regains its function in less than five days. This test, he claims, is of value in nephritis complicating pregnancy.

Our findings apparently differ markedly from those obtained by Clauser, and moreover, unlike him, we do not regard the depressed thiosulphate output during pregnancy as necessarily being evidence of a disordered renal function. Neither do we consider that it can be explained in terms of hepatic insufficiency. At this stage of the investigation we are forced simply to draw attention to the fact that in most cases of pregnancy the thiosulphate metabolism is disturbed to a varying degree.

Comparison with Findings in the Laboratory Animal.

Pregnancy in the bitch differs from human pregnancy in several respects. Its duration is about one-fourth of that in the human being, the pregnancy is usually multiple, and the rate at which foetal tissue is laid down is relatively very much greater than that which obtains in the human subject. These considerations may help to explain the fact that while the thiosulphate depression during pregnancy in the bitch was consistently found (in a small series) and always well-marked, that which occurs in human pregnancy is less consistent and frequently of minor degree.

In view of these differences, the possibility of a diagnostic application of the test, which was suggested by the experimental findings, has not been realized, since a markedly low thiosulphate excretion, which we still consider in the absence of renal deficiency to be strongly suggestive of pregnancy, is not encountered till the stage when pregnancy can be more simply and more positively diagnosed by clinical examination.

As already mentioned, human pregnancy complicated by toxæmia or nephritis will be dealt with elsewhere.

SUMMARY.

1. The excretion of intravenously injected sodium thiosulphate solutions containing approximately 0.95 grammes, 1.8 grammes and 2.6 grammes of the crystalline salt has been studied in a series of

non-pregnant and pregnant patients suffering from venereal disease.

2. With the exception of a small group of complicated cases the thiosulphate excretion of the non-pregnant group of patients fell within normal limits. The percentage excretion was found to increase with increasing amounts of sodium thiosulphate injected.

3. In almost all cases the percentage thiosulphate excretion was found to be depressed to a varying extent below the normal value during the latter part of pregnancy and also in many cases during and after the puerperium, the patients falling into two main groups according to the extent of the depression.

4. In the first group the depression was not extensive and was of short duration, while in the second group the depression was marked and prolonged, persisting at times up till twelve months after confinement.

5. None of the patients studied showed any clinical evidence either of renal insufficiency or of toxæmia of pregnancy.

ACKNOWLEDGEMENTS.

We wish to express our sincere thanks to Dr. Charles A. Frew, Honorary Assistant Physician for the treatment of Female Venereal Diseases, Royal Prince Alfred Hospital, for affording us free access to the patients under his care, and to Dr. F. N. Chenall and Dr. C. H. McDermott, who during the period of this investigation successively occupied the position of Venereal Diseases Registrar, and the hospital staff, for their sympathetic co-operation.

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THE ORIGIN OF SLUDER'S OR SPHENO-PALATINE NEURALGIA.

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IN 1908 Sluder, of St. Louis,⁽¹⁾ described a type of neuralgia which he believed to be associated with infection of the nasal or spheno-palatine ganglion or its related branches. His most recent views were elaborated in a volume published in 1927 prior to his death.⁽²⁾ It is named by him nasal or spheno-palatine ganglion neurosis, or "lower half headache."

The accuracy of his clinical description of this syndrome must be generally admitted, as also must his view that it occurs after infection of the nasal cavity and paranasal sinuses and, in particular, the sphenoidal and posterior ethmoidal group. On the other hand, the relief of the pain by alcohol coagulation in the vicinity of the spheno-palatine ganglion has been disappointing in Melbourne, and a criticism of Sluder's views is here offered with an alternative method of treatment.

Anatomical Relationships.

To appreciate Sluder's views on this peculiar and not uncommon type of neuralgia, the relationships of the sphenoid bone must be considered in some detail.

At birth the formation of bony sinuses of the head is very little in evidence. In the newly born child the maxillary and mastoid antra, and usually a few mastoid cells, are already formed, but the frontal, sphenoidal and ethmoidal sinuses are represented by mere pits in appropriate positions in the nasal cavity. The curious process of enlargement or pneumatization then begins to take place and progresses until death.⁽³⁾ What controls this process is not known, but a radiological examination of an acromegalic at present under observation reveals gross pneumatization associated with the typical appearances of the *sellae turcicae* in this condition. This process of pneumatization may not be confined to the body of the bone, but may invade the wings or the pterygoid plates. Hence the sphenoidal cavity must at times become intimately related to the important nervous and other structures that the sphenoid bone transmits or is adjacent to; the possibility of their infection by sphenoidal sinus suppuration must be seriously considered. The posterior ethmoidal cells, although originating from a different nasal area, cannot be dissociated from the sphenoidal cells, as they themselves are adjacent and are frequently concurrently infected; they may pneumatize the lateral part of the body of the sphenoid, a point that the surgeon operating on this area must always bear in mind.

The structures which may share in sphenoidal sinus infection are as follows:

1. The optic nerve is frequently in contact with a lateral cell, hence the clinical association of retrobulbar neuritis with sphenoidal sinus infection.

2. The pterygoid canal containing the Vidian nerve lies on the floor of the sinus and is described by Sluder as frequently standing out like an aqueduct of old, to divide the cavity into two lacunae. The bony wall is also not infrequently absent. The importance which Sluder gives to this small nerve will be appreciated later, when his thesis is considered.

3. The *foramen rotundum* is not infrequently in contact with air cells and may be surrounded by them.

4. If the great wing of the sphenoid is pneumatized, the mandibular division of the trigeminal nerve in the *foramen ovale* is occasionally in contact with air cells, and if the process is extreme, the cells may even come into close relationship to the Gasserian ganglion itself. This is very unusual, but Sluder shows illustrations of a few specimens clearly demonstrating the possibility.

5. The orbital fissure might also be encroached upon, but this fissure is wide and the structures within loosely held.

6. The cavernous sinus and its contained structures are also adjacent; cavernous sinus thrombosis is a well known clinical condition. Involvement of the various ocular nerves is clinically known and it is not impossible that the ophthalmic nerve should be occasionally infected by this route.

7. Another relationship of importance in Sluder's thesis is that of the spheno-palatine ganglion. The pterygoid plate forms its posterior relationship, and though this is usually solid, it may occasionally be pneumatized.

Bearing in mind the various possibilities of the spread of sphenoidal sinus infection to adjacent parts, it may be as well before further considering Sluder's views to refer to the various branches of the spheno-palatine ganglion and the nerve supply of the sphenoidal and ethmoidal sinuses in relation to the actual syndrome.

The innervation of these structures is entirely from the trigeminal nerve. Briefly, the lower and main part of the sphenoidal sinus, with the posterior ethmoidal cells, is supplied by branches from the second division of the fifth cranial nerve by way of the spheno-palatine ganglion, while the upper part derives a few fibres from the first division by way of the anterior ethmoidal nerve in its intracranial course. This nerve passes on to innervate among other things the anterior and middle ethmoidal cell groups.⁽⁴⁾ The point must therefore be borne in mind that these cells derive their nerve supply from both upper divisions of the trigeminal nerve.

Symptoms of Disease of the Sinuses.

Disease of each of the various paranasal sinuses has its own symptomatology.

The particular syndrome of sphenoidal empyema is pain in and about the eye, extending down over the cheek to the upper teeth, and back into the temporal region. There is usually, as well, a duller pain referred to the back of the head and to the mastoid process; its maximum area of tenderness

is situated somewhere about 5.0 centimetres (two inches) directly posterior to this process. The pain may even extend down the neck and into the shoulder and, in extreme cases, into the hand. It must be noted that this is the sphenoidal syndrome in its fully manifest form. It is not always present as such and parts only of this area of distribution of pain may be affected. In particular cases the area of pain may be so altered as to mimic each type of paranasal sinus inflammation and be discriminated only by careful examination and analysis.

Operative treatment on the sphenoidal sinus and adequate drainage usually result in relief of pain. On the other hand, even with the most careful and apparently successful treatment, relief may not occur; the condition of neuralgia described by Sluder may then persist over the whole or some part of the area typically affected.

The quality of the pain is more that of a neuralgia than a headache, yet it has many essential differences from the usual "tic douloureux" of fifth nerve origin. It is more constant in its nature and does not show the cataclysmic stabs of the true tic. It is not usually quite so severe, but more continuous and nerve racking. It may have remissions, but when once established it is extremely persistent. Again, unlike the true tic, it is not influenced by mastication or by hot or cold fluids, nor are there any "trigger points" associated with it, although the pain may be increased by pressure over certain areas. It is also apt to occur in younger patients.

Sluder named the condition "lower half headache." The occipital pain seems to approach more to the quality of "headache" than the anterior pain, while the pain in the neck and shoulder is often described as "rheumatically" in type.

Sluder's view of the causation of this syndrome is that it is due to irritation of the small sphenopalatine ganglion or its branches, and the cure lies in alcohol coagulation of this ganglion.

The Sphenopalatine Ganglion.

The sphenopalatine ganglion lies deeply placed in the spheno-maxillary fissure, below the maxillary division of the fifth nerve, from which it is, as it were, suspended by several small communicating branches. It is fortunately fairly easily accessible by way of the nose, as it lies rarely more than 4.0 millimetres in depth, lateral to the large sphenopalatine foramen. It is here readily accessible for injection with cocaine solution and, incidentally, open to infection. Sluder's method of alcohol injection is clearly described in his book.⁽³⁾

The actual ganglionic part is small and consists of a few small stellate cells in an intricate mass of nerve fibres.

To appreciate Sluder's thesis, it is of great importance to understand the components of the various roots and as to whether these actually anastomose with the ganglionic cells or are merely birds of passage gathered into this area by the process of development prior to passing elsewhere.

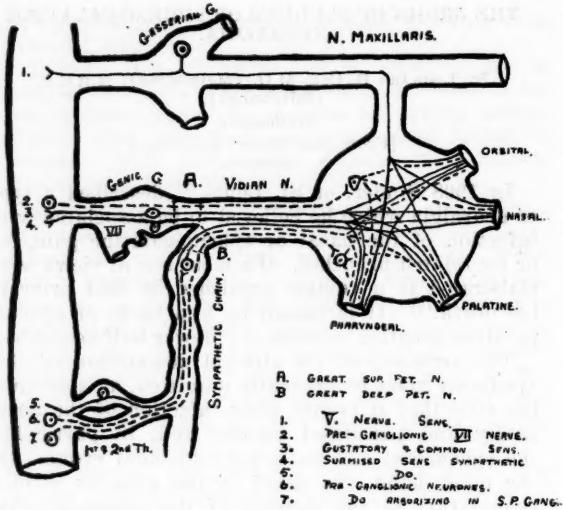


FIGURE I.
Connexions of the Sphenopalatine Ganglion
(after Sluder).

Three roots are described, all of which are in reality mixed nerves (see Figure I).

The Motor Root.

The "motor root," so called, is in the main the Vidian nerve. This, it will be remembered, lies in the floor of the sphenoidal sinus and is liable to infection there.

The major portion of the motor root consists of autonomic preganglionic fibres from the medulla by way of the facial nerve, which arborize about the stellate cells of the sphenopalatine ganglion. These supply glands and unstriped muscle in the region of the palate.⁽⁷⁾ The motor root also contains sensory fibres which come by way of the ganglion from the soft palate and adjacent parts of the pharynx to the geniculate ganglion of the facial nerve. Sluder⁽⁴⁾ quotes Schaefer in saying that there is supporting evidence of "sympathetic" sensory fibres, whose ganglion is the geniculate ganglion and which conduct sensation from a wide distribution in the nasal cavity.

In passing it may be said that the importance of the Vidian nerve lies in the fact that Sluder attributes the posterior pain of the syndrome to its irritation.

The Sympathetic Root.

The sympathetic root is from the great deep petrosal nerve, which is in a sense the continuation of the carotid plexus. It consists of post-ganglionic fibres from the superior cervical sympathetic ganglion, but Sluder believes that there are also pre-ganglionic fibres in this root as well as sensory fibres passing to the upper thoracic roots.

The Sensory Root.

The sensory root consists of two or three short branches from the maxillary nerve which pass by way of the ganglion area to ethmoidal and

sphenoidal air cells to a large part of the mucous membrane of the nose and to palate and gums. Here, then, we have, following this route, but in no way associated with the nerve cells of the ganglion, the sensory nerve supply of a large part of the mucous membrane of the nose and the ethmoidal and sphenoidal sinuses.

The Basis for Sluder's Theory.

Sluder's theory of the origin of the syndrome is based on the following points: (i) Cocainization of the mucous membrane of the nose adjacent to the sphenopalatine foramen has relieved the pain; alcohol coagulation of the ganglion by this route has cured it. (ii) Cocainization of the floor of the sphenoidal sinus, and thus the Vidian nerve, relieves the occipital pain, while the application of a Faradic current to this area produces pain in the occiput, neck and shoulder.

The facial and ocular pain is therefore produced by stimulation of the branches from the maxillary nerve, the radiating pain at the occiput by irritation of sensory fibres associated with the involuntary nervous system whose sensory nucleus is the geniculocate ganglion of the seventh cranial nerve. How this causes radiation to the upper cervical segments can be imagined.

The Nature of Sluder's Neuralgia.

In addition, however, to the cure of this type of headache by treatment directed to the sphenopalatine ganglion, we read of the cure of a multiplicity of conditions. Some such results are understandable, as the relief of all eye pains, asthma, spasmodic contraction of the gullet, migraine and fibrositic pains of the neck and shoulder, but when we read of the accidental production of sciatica and the relief of lumbago and the pains caused by uterine carcinoma it gives us cause to think.

Sluder admits that in some instances the neuralgia remains unrelieved by cocainization of the ganglion. The infection then, according to him, must be more centrally placed, and cocainization of the sphenoidal sinus, hence the Vidian nerve, will usually give relief. The Vidian nerve has become infected as it lies on the floor of the sinus and the maxillary nerve by a spread of infection to the region of the *foramen rotundum*. Pain in both anterior and posterior areas is produced by this dual infection. But some patients are unrelieved after either of these measures. Sluder does not seem to have considered the question of alcoholic coagulation of the Gasserian ganglion by way of the *foramen ovale* for the relief of those in whom other measures have failed.

The following report of a case is made in considerable detail, as the intelligence, cooperation and endurance of the patient enabled her condition to be carefully studied, although, as shown, the cause and the final good result in no way bore out Sluder's theories of the causation of pain in this neuralgia.

The patient, a highly intelligent woman of twenty-eight years, was seized in April, 1926, with a right-sided pain

of the Sluder type over the right eye, cheek, temple and occiput associated with a rise in temperature. X ray examination revealed a solitary empyema of the right sphenoidal sinus, which was considered possibly a blood-borne infection, as the remainder of the nasopharynx and accessory sinuses were quite normal. Following operation by Mr. G. C. Scantlebury, there was only partial relief of symptoms, and although she was repeatedly examined by means of X rays no further evidence of infection was found. The opening into the sphenoidal sinus was sufficiently large to allow direct observation and to permit intrasphenoidal treatment and experiment. The typical full syndrome of Sluder's neuralgia persisted, with intermissions, for two years, until in 1928 it became very severe and seemed each month to increase in intensity, spreading little by little down the neck and into the shoulder. Fortunately the anterior pain could be objectively delimited by a persistent and invariable hyperesthesia. The occipital and shoulder pain could not be so marked out, but could be tested by the subjective tenderness to light pressure, but the most exquisitely sensitive part of all was the right eyeball, pressure over which seemed to cause intolerable pain. The method of delimitation was that employed by Head⁽¹⁾ in his classical work on hyperesthesia of the head and face in 1894.

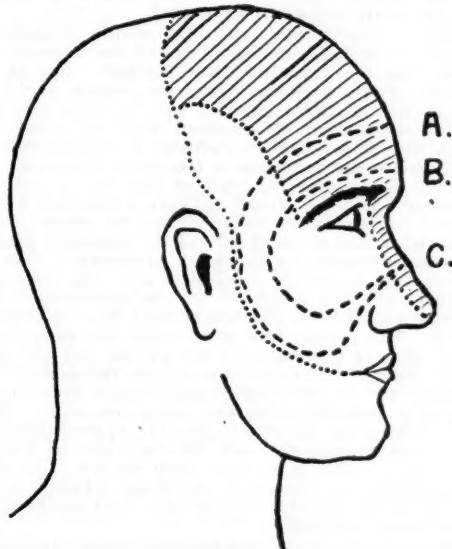


FIGURE II.

Showing conditions before and after cocainization.
 A to C = area of hyperesthesia before cocainization.
 B to C = area of hyperesthesia after cocainization.
 The shaded area represents the area supplied by the ophthalmic division of the trigeminal nerve, the area enclosed by dotted lines represents the area supplied by the maxillary division.

After the patient had undergone a long course of medical treatment, general and intrasphenoidal, without result, Mr. Scantlebury, in September, 1928, following Sluder's technique, cocainized the nasal mucous membrane in the region of the right sphenopalatine foramen. A slight lessening of the anterior pain was noticed, particularly in the upper teeth. There was an objective contraction of hyperesthesia and, although this was not mapped out during this experiment, the contraction was general and particularly well marked in the temporal area. Cocainization of the floor of the sphenoidal sinus was performed almost coincidentally. Shortly after this the occipital pain disappeared and with it the tenderness posterior to the mastoid process, but in about ten minutes it reappeared and gradually increased in intensity. It was not quite clear that this loss of pain might not have been a later effect from cocainization of the lateral wall of the nose. Some days later the experiment was repeated; cocainiza-

tion about the region of the spheno-palatine foramen resulted in a concentric retraction of hyperesthesia which was mapped out (see Figure II). It was not nearly so pronounced, subjectively or objectively, as in the first experiment. Again the sensation in the upper teeth was the first to disappear, but the occipital pain was stated to be rather intensified than otherwise. At the end of about forty-five minutes the floor of the sphenoidal sinus was cocainized. A slight diminution of occipital pain was said to occur. Although even a wider area than Sluder advocates for cocainization was employed, the neuralgia could certainly not be said to have disappeared, and the relief was slight. As, however, there had been some diminution of pain, it was thought advisable to attempt alcohol coagulation of the spheno-palatine ganglion, following Sluders' technique.

Four attempts at this were made by Mr. Scantlebury; fairly large quantities of alcohol were injected, but no relief was experienced, although on one occasion there was a slight diminution of sensation over the cheek. This corresponded to a small triangle of hypoesthesia in the area usually associated with the maxillary division of the fifth nerve; it was probably due to soakage into this nerve.

In January, 1929, Dr. Julian Smith injected the various superficial foramina over the whole of the hyperesthetic area, but although there was a deep supraorbital anaesthesia, no symptomatic relief occurred.

Finally, a few weeks later, Dr. Smith attempted to inject the Gasserian ganglion by way of the *foramen ovale*. Wilfred Harris's⁽¹⁰⁾ method was carefully studied and followed. The mandibular nerve was struck and a few drops of cocaine were injected into it with the usual result of diminution of sensation over the area of its distribution. The needle was pressed a little further into the *foramen ovale* and again a few drops of cocaine were instilled. The immediate effect of this was a feeling of "sandiness" about the eyeball and then a diminution of the headache. It seemed likely then that, as pointed out by Harris, the medial group of cells of the Gasserian ganglion was being infiltrated by the local anaesthetic. This particular group is mainly associated with the ophthalmic division of the fifth cranial nerve. As symptomatic relief was being experienced, absolute alcohol was injected drop by drop. The patient was seized with the most intense burning pain in the region of the eyeball and she stated that the ordinary neuralgia was grossly intensified. Soon afterwards the occipital and shoulder pain began to disappear and, coincidentally, the entire neuralgia. There was no subsequent anaesthesia, but the hyperesthesia had entirely disappeared, although the right eyeball was still slightly more tender to pressure than the left. The symptomatic result was dramatic; morphine, which had been taken in amounts up to 0.13 grammes (two grains) daily, was no longer necessary.

In six months' time the pain had reappeared. An identical injection was performed, with an identical result, but again the amount of alcohol injected was small and not sufficient to produce anaesthesia. Owing to the easy accessibility of the *foramen ovale* in this patient, general anaesthesia was employed on this occasion. To date, over a period of two years, there has been no recurrence of the pain.

It is evident, then, that Sluder's neuralgia in its complete form is curable by the same measures that are employed for the treatment of ordinary major trigeminal neuralgia. It may therefore be surmised to be a definite clinical variety produced by a septic process in a somewhat different area in the trigeminal distribution from that in which the major tic occurs.

In considering Sluder's views, that the syndrome is a manifestation of irritation of the spheno-palatine ganglion, with a posterior pain as a result of irritation of the Vidian nerve somewhere on its course, it should be noted that this ganglion is a pure cranial autonomic ganglion, and no irritation

of it alone could produce other than secretory or motor symptoms. As branches of the second division of the fifth cranial nerve pass alongside or intermingle with this ganglion on their way to the nasal cavity and sphenoidal and ethmoidal sinuses, there is no reason why an injection of alcohol into this region should not relieve the symptoms of this neuralgia, provided that the source of irritation is still peripheral to this area and in those branches from the second division rather than the first division of the fifth cranial nerve. It must also be remembered that there is such a thing as "nerve shock." Harris has frequently noted cure of pain in the eyeball following injection into an unrelated nerve, the infraorbital nerve.

In the light of Sluder's experience, especially as his operation is not particularly difficult and is comparatively harmless, it is still likely to remain the first method of injection for this condition, particularly as many of the patients are undergoing treatment by rhinologists, to whom the technique is comparatively simple.

Sluder's views of the origin of the posterior pain are open to criticism. He admits that in cocainization of the sphenoidal sinuses at times he has produced anaesthesia of all three branches of the trigeminal nerve. The relief that he attributes to cocainization of the sensory fibres of the Vidian nerve may in reality be some effect either on the branches of the fifth nerve or even on the medial portion of the Gasserian ganglion, if pneumatization is extreme.

The Vidian nerve undoubtedly carries sensory fibres to the geniculate ganglion of the facial nerve, fibres which are thought in part to be gustatory in function. Herpes of the geniculate ganglion, however, produces neither vesiculation nor pain in the occipital region. The distribution is more before and above the ear and at the back of the lower jaw. The vesicles which sometimes occur on the soft palate and the corresponding half of the tongue seem more likely to represent the area of distribution of the sensory fibres in the Vidian nerve. It is quite possible that there are other sensory fibres associated with the autonomic nervous system, but it does not seem necessary to assume their presence to explain the radiation of the pain to the cervical and upper thoracic nerve areas. In the patient studied the occipital pain was almost certainly a radiation from the fifth nerve, by way of its descending root, to the upper cervical segments, gradually extending to the more caudal segments as the pain increased in severity.

This radiation of frontal pain to the occiput is of everyday clinical occurrence. By reference to the anaesthesia produced by syringo-bulbia of the descending root of the fifth nerve, it is seen that the most caudal lesion is associated with disturbance of sensation in the frontal area.⁽¹⁰⁾ This area, then, is supplied from a section of the central nervous system next in anatomical sequence to the first and second cervical segments from which the occiput receives its sensory innervation. Even intracranially

the recurrent meningeal branch of the ophthalmic division of the fifth cranial nerve passes dorsally to the tentorium and anastomoses with the intracranial branches of the upper cervical and lower cranial nerves in the posterior fossa of the skull. The radiation of a frontal type of pain to the occiput should not then come as a surprise.

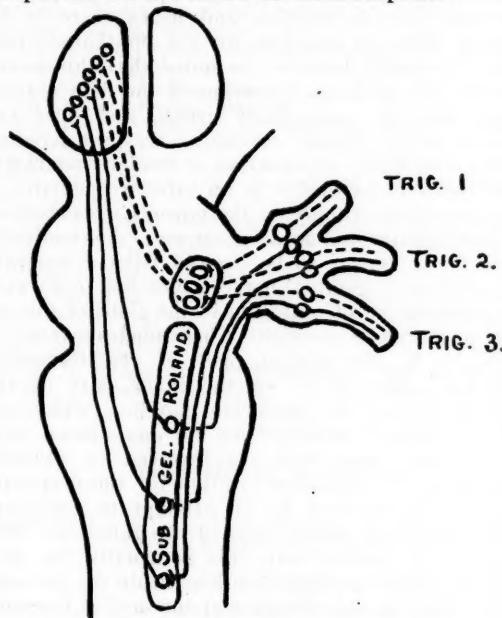


FIGURE III.

Showing the relationship of the fibres carrying protopathic sensation in the fifth cranial nerve to each other and to the *substantia gelatinosa* (after Bing).

Sluder's Neuralgia and Tic Douloureux.

What, then, is the fundamental difference between Sluder's neuralgia and the ordinary major tic?

The rarity of a *tic douloureux* confined to or starting in the first division of the fifth cranial nerve has been frequently commented on. The usual exception is supraorbital neuralgia. This nerve, in addition to the skin, supplies the mucous membrane of the frontal sinus; this explains the well known frontal distribution of the pain of frontal sinusitis. A true supraorbital neuralgia, however, may also result from frontal sinusitis. It is usually easily curable by injection into the supraorbital foramen, so that here we have a peripheral neuralgia with a peripheral cure.

Sluder also describes an anterior ethmoidal nerve neuralgia in which the pain is situated between the eyebrows. It would appear to be a peripheral condition confined to the anterior ethmoidal nerve, for it is said to be easily relieved by cocaineization of this nerve at its entrance into the nose.

The major tic results from irritation of the second or third division of the fifth cranial nerve and, save that it is sometimes due to disease of the maxillary antra, it is not commonly of sinus origin. Reference to the area of hyperesthesia of Sluder's neuralgia

(see Figure II) shows that cutaneous areas supplied by both first and second divisions have become hyperesthetic; the pain in the eyeball strongly suggests a considerable involvement of the first division.

As has been previously noted, the sphenoidal sinus and posterior ethmoidal cells derive their sensory supply from both of the upper trigeminal divisions. The first division, by way of its naso-ciliary branch, not only supplies these cells, but is the great sensory nerve to the globe of the eye and, in addition, carries preganglionic fibres to the ciliary ganglion. By its anterior ethmoidal branches it supplies both anterior and middle ethmoidal cells, part of the septum and the lateral wall of the nose. The second division, by its sphenopalatine branches, which pass adjacent to or interwoven with the fibres of the sphenopalatine ganglion, supplies the sphenoidal and posterior cells and the lateral wall and septum of the nose. Thus the nasal cavity and its accessory sinus have a dual nerve supply.

It is obvious that the distinction between Sluder's neuralgia and ordinary trigeminal neuralgia cannot merely be a difference of involvement of the first division as opposed to the involvement of the second or third division of the trigeminal nerve. The hyperesthesia is neither confined alone to the first division nor does it involve the whole of the cutaneous area supplied by it. It will be noted that supraorbital and supratrochlear branches are relatively little concerned. The neuralgia corresponds in fact to no group of cutaneous nerves and can only be due to the sensitization of some actual cell group by irritation of other deeper fibres whose connexions are related thereto.

This phenomenon of superficial hyperesthesia is of common occurrence clinically, as in the hyperesthesia of the chest wall in *angina pectoris* or the abdominal hyperesthesia of appendicitis or renal colic.

Neuralgia of the naso-ciliary nerve could simulate much of Sluder's syndrome, but hardly the hyperesthesia of the second division of the trigeminal nerve. If the sphenopalatine nerves were involved as well as the naso-ciliary nerve, the picture should be more complete, as both first and second divisions would then be represented.

As these nasal and ocular nerves have no cutaneous branches, with the exception of the small terminal branch of the naso-ciliary nerve, the question arises as to whether the trigeminal nerve may not have a double function and these nasal and ocular fibres, nuclear and central connexions, somewhat set apart from the cutaneous and dental fibres. A study of comparative anatomy reveals that phylogenetically this condition actually exists. Gaskell pointed out that the trigeminal nerve is a conglomeration of the sensory roots of all the cranial segments. We may then expect it to be mixed in its function. In cyclostomes and fishes⁽¹¹⁾ it is a nerve of two portions only (see Figure IV).

1. The anterior portion is the deep ophthalmic nerve which runs in the medial wall of the orbit.

It is the nerve of the ethmoidal region and in man is represented by the naso-ciliary nerve.

2. The posterior portion supplies the first visceral arch and its musculature. The post-trematic branch represents the mandibular nerve and is motor and sensory. The pretrematic branch (*ramus maxillaris*) is purely sensory and supplies the skin and mucous membrane of the superior maxilla. A superficial branch from this runs above the eye and forms in the human the ophthalmic nerve.

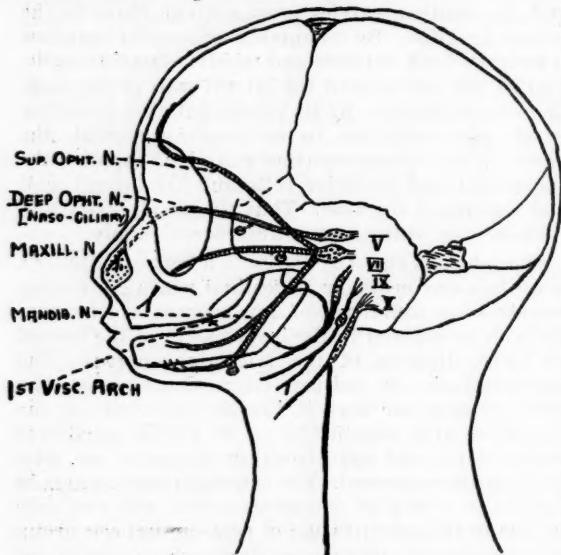


FIGURE IV.

Showing the distinction between the deep ophthalmic nerve from the remainder of the trigeminal nerve (after Lubosch).

The duality of function of the trigeminal nerve in these early forms can thus be clearly recognized, one part being associated with the ocular and olfactory apparatus, the other with the structures derived from the first visceral arch. This distinction is of great interest when we consider the distinction between *tic dououreux* and Sluder's neuralgia. Phylogenetically, the supraorbital and other cutaneous branches of the first division of the trigeminal nerve are derived from the second division; radiation of pain to these branches is thus not uncommon in the major neuralgia of the second division. On the other hand, the naso-ciliary nerve is a separate anatomical entity, fused with the nerve of the first branchial cleft; it may then be surmised that the sphenopalatine branches of the second division may belong to this same sensory system.

It is suggested that the cell group which has to do with this naso-ciliary system, and hence Sluder's neuralgia, may be mainly in the medial part of the Gasserian ganglion, that is, the part largely associated with the ophthalmic nerve or the first division. Frazier and Whitehead⁽¹²⁾ noted the distinctive development of the ophthalmic group of cells in the Gasserian ganglion of the human embryo.

Their inspiration for this work was the effect of subtotal resection of the sensory root for the relief of *tic dououreux*. They found that the neuralgia was cured if all but the two inner fasciculi of the sensory root were resected, while the risk of keratitis was avoided. The ophthalmic group in their observation was thus a distinctive part. They point out also that in reptiles and fishes there is an entirely different ganglion for the ophthalmic portion. It should, however, be noted that this is not strictly the ophthalmic portion of the fifth cranial nerve, but the naso-ciliary group, and does not contain the cutaneous branches of the first division.

The most likely explanation of Sluder's neuralgia then seems to be that it is an infective neuritis of the nerve fibres supplying the mucous membrane of the nasal cavity and adjacent sinuses. Anatomically these fibres appear to be derived from a separate entity in the trigeminal nerve, which has to do with the appreciation of sensation in the globe of the eye and in the nasal cavities. Their nuclear group is probably in the medial part of the Gasserian ganglion. The fibres on the inner part of the sensory root of the Gasserian ganglion, which are left in subtotal resection for *tic dououreux*, may be the very ones that are involved in Sluder's neuralgia. The simplest solution for the desperate case would therefore be an attempt to coagulate with alcohol the medial part of the ganglion. Fortunately, as pointed out, this is usually the first part affected by alcohol injection within the *foramen ovale*. Failing this, division of the medial fasciculi of the sensory root would be most likely to give relief.

There is a variety of conditions of paroxysmal or persistent pain about the eyeball, extremely puzzling to the clinician and most nerve racking to the patient. Sometimes these conditions seem to be of the nature of a simple neuralgia of the eye, but in other cases they border on migraine or what is described as migrainous neuralgia, owing to the association with nausea and vomiting. The term "ciliary neuralgia" is used by some authors for pain confined to the eyeball. Frequently these patients are unrelieved by the most careful attention of oculist and rhinologist and no sedative prescribed by the physician does more than give temporary relief. Skiagraphy of the nasal sinuses may reveal no abnormality or, if an inflammatory condition be found, operation brings no ease. The pain is often very similar to that of Sluder's neuralgia, and it is not improbable that some of these conditions may be actually due to infective neuritis of naso-ciliary or sphenopalatine nerves. When we consider the area of orbit, nasal cavity and sinuses supplied by these nerves we realize what a very extensive area there is here for the onset of a possible primary infection.

Eye strain, imperfect muscle balance, errors of refraction might all cause pain similar to that of Sluder's neuralgia, but should be relieved by appropriate correction. Neuralgia due to an inflammatory condition of the eye might, however, persist unexplained, though the primary cause be eliminated.

The nasal cavities, with their poor drainage and constant liability to coryza, could be a fruitful source of pain. In many of these cases the question of treatment directed along the lines indicated in Sluder's neuralgia should be seriously considered.

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Reports of Cases.

SUGGESTED SURGICAL TREATMENT OF BONE IN RHEUMATOID ARTHRITIS.

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THROUGH the accidental fracturing of the femur of an elderly lady, the victim of rheumatoid arthritis, my attention has been directed to a means of treatment of this condition.

The case is that of Miss L., an elderly lady, who has suffered for fifteen years from rheumatoid arthritis. She has typical hand and finger deformity and is practically crippled owing to the condition of her knees. I had treated her previously for another condition not associated. In April, 1930, whilst trying to make herself useful in the kitchen of her home, she fell and fractured her femur. X ray examination revealed an extracapsular fracture of the neck of the left femur with a splitting of a large fragment of the great trochanter. The patient was very anaemic and frail and very helpless. She had been unable to put her hands behind her back for thirteen years. She was put into a nursing home, where she remained for eight weeks.

Owing to her age and the state of her knee, no attempt at treatment by fixed apparatus was made. Her affected limb was placed on pillows and cocked up into an elevated abducted position.

From the time of her admission to hospital after the first results of shock and pain had worn off, and this was within a few days, she had a most interestingly easy time. She slept well, ate well, and generally thrived. The old torturing pains which she had suffered for many years left her. Her general health improved in the most extraordinary way. An anaemic condition, almost amounting to cachexia, improved and she developed a good, bright colour. An old eye condition, iritis and conjunctivitis, which had troubled her for years, cleared off and she became, in her own words, "a new woman."

She got perfect union and function in the left hip and her knees improved most strikingly. She was able to get about better than she had for years and could meet her hands behind her back.

Apart from the improvement to be expected from rest, nursing and perhaps more regular diet, it seems undoubtedly that something had done her an immense amount of good. This leads one to the thought that the fact of fracturing the bone, with the resultant releasing or freeing of substances, produced favourable effect on the whole system. Thinking along these lines brings one to the point where a revision of pathological teaching may be necessary. Study of all the literature on the pathology of this condition would lead us to believe that the change is always a primary joint change and that changes in bone follow. I cannot find any discussion of whether the change is primary bone or primary joint, except in another type which is described as a true osteoarthritis—the type where lipping of bone margin with cartilaginous erosion occurs—*morbus coxae senilis*, the hip joint being the favourite joint, as occurring in elderly people.

Pathology.

Muir, in his text book on pathology, in dealing with rheumatoid arthritis *adhesiva chronica* writes:

When these changes are going on in the joint, atrophic processes occur in the bone with resulting rarefaction or porosity.

The results of treatment are so unsatisfactory that one is more than persuaded to take a look from another angle. Let us argue that the condition is definitely a primary bone condition. Does this fit in with the symptomatology and progress? One can say "Yes." Does not the disease set in with pains along the limbs, stiffness or muscle numbness? Could not these pains be wholly and solely due to bony inflammation?

This is, I think, correct, and we have all seen these cases running their horrible course. Do X rays help? Little or not at all. There may be the grossest bony change, absorption of shaft of long bones amounting almost to scooping out, absorption and rarefaction of cancellous tissue at both ends, with a preservation of the outer layers which deceives by its apparent density and gives no idea of the condition within.

Prompted by the results of the accidental fracture in the case of Miss L., I determined in suitable cases to imitate the results of fracture, and thinking of the best means, I thought that trephining and punching large openings in the bone without disturbing continuity would be of use.

The first patient I selected, a lad, G.B., *ad statis* eighteen, who for three years had progressed from bad to worse despite all treatment, rest, vaccine, in hospital and out, had both knees and hips involved, flexion of knees, flexion and adduction of hips.

Cutting down on the lower end of each femur anteriorly, I proceeded to trephine out a piece and open up the bone. The trephine on each bone simply fell into the medullary cavity from which, under pressure, yellow medullary oil exuded. The bone was no thicker than the wood in a cigar box and one could have pushed a finger through it. The cancellous tissue was so frail that one could swab it out. He suffered no disturbance from this and in fact said he felt better. At later stages I opened the upper end of the femur on both limbs and the upper ends of both tibiae. I also divided his hamstrings to try to overcome knee flexion. Whilst he was on the table preparatory to division of his hamstrings, he made a slight movement and fractured his right femur through the lower third. I did not divide his thigh adductors, but just stretched them. Prior to this he had, since the first operation, been able to separate his knees three or four inches for the first time for months. His general condition had improved immensely and he expressed himself as greatly improved and quite free from pain.

I have put him on various bone-forming preparations, "Tricalcine" and adrenalin, and have sent him for six weeks to Caulfield Hospital to convalesce.

Since I began to pursue this line of investigation, I have heard of odd cases in which great improvement has followed fracture. Fracture resulting from attempts to reduce deformity has also seemed to cause improvement.

My reason for writing this note is to suggest to others that they may think likewise. They may remember cases in which improvement after accidental fracture has

occurred out of all proportion to the improvement to be expected from the necessary rest and nursing.

My view is that in early cases drilling all affected bones early will help. Do not wait for deformity and helplessness. I propose to neglect the joint, taking it that this is secondary. This may seem presumptuous, and for all I know may have been tried and found useless, but undoubtedly I have had some encouragement and shall be glad to know the result of investigation by colleagues.

Reviews.

HYGIENE.

The issue of the seventh edition, completely revised and brought into line with modern conceptions of preventive medicine, stamps "A Treatise on Hygiene and Public Health, with Special Reference to the Tropics," by Birendra Nath Ghosh, as a text book not only justly popular, particularly with students in India, but as a work which takes all preventive medicine as its province.¹ It can be commended generally to all teachers and students of public health.

The book has been practically rewritten with the assistance of Lieutenant-Colonel A. D. Stewart, Professor of Hygiene, Calcutta School of Tropical Medicine, and revised by his predecessor, Colonel A. B. Fry.

There is not only the usual information given under "Water," "Air," "Ventilation" and the other headings of the ordinary text book, but there are many little details showing a wide, general and up-to-date knowledge of the subject. Practical application of general principles are given in definite instances, such as the general duties of the municipal authorities in respect to the supply of water under the *Calcutta Municipal Act*, 1913. Especially excellent is the description of the artificial purification of water, including chlorination and the control of filters.

Under "Ventilation" the stimulating effect of moving air upon the skin, dependent on the evaporating power of the air, is emphasized, as also is the artificial cooling of air during the summer by fan wheels through wet *Khus Khus* mats or *Tatties*, which also act as filters for suspended impurities.

There is a useful chapter on occupational hygiene and offensive trades. In view of the forty-four hours' week in Australia, it is interesting to note that under the Indian *Factories Act*: "No person should be employed in a factory for more than sixty hours in any one week and for more than eleven hours in any one day."

The chapter on soil, houses and buildings, although adapted to Indian conditions, should be of particular interest to those dwelling in the tropical portions of Australia and her mandated territories.

Food, diet in India, vegetable foods, animal foods, beverages and condiments are dealt with in an interesting way by which many useful facts not usually given in text books on hygiene are noted.

One of the best and most informative chapters of the book is that dealing with the disposal of human excreta. The details given of the various types of latrine and method of laying out trenching grounds make this work of value to military as well as civil sanitarians who have to supervise or control conservancy systems.

The Eastern type of latrine, with the squatting plates with two foot rests on either side of the pan, placed on a level with the floor, if adopted generally in public conveniences, would remove obvious objections and secure a natural position in defecation.

There is an excellent description of a septic tank installation, as well as of an activated sludge plant.

With regard to insecticides, kerosene or petrol mixed with some essential oil, such as oil of sassafras or

eucalyptus or tar oil, has evidently proved as effective in India as it did in Sydney during the last outbreak of plague.

The chapters on malaria and mosquitoes, rewritten by Major Covell, of the Malaria Survey of India, are the best summary of the subject we have seen in any recent text book. In a future edition it is suggested, however, that the tabulated duties of mosquito brigade men from Major James's "Malarial Fevers" be, as in the previous edition again included as being especially useful for teaching routine procedure.

The Lelean Sack Disinfector, a light, simple apparatus for disinfecting clothing and bedding by current steam, is well described and illustrated.

The "Sanitation of Fairs and Religious Festivals" affords evidence of the special problem of sanitation in the East.

The final chapter on vital statistics, still further illustrates how much valuable knowledge is to be found in this well of hygiene.

A text book such as this makes obvious the need for a similar compendium adapted to the requirements of students of hygiene in Australia.

DIETETICS.

"DIET IN DISEASE," by George A. Harrop, Junior, is a book that can be highly recommended to physicians and particularly to all interested in the question of diet.² The book is thoroughly up to date and is compiled in a most interesting way. It is divided into three parts.

Part I deals with the requirements of nutrition and is further subdivided into four chapters which deal with the energy requirement, the protein requirement, the mineral and fluid requirement and, lastly, the vitamins. These are thoroughly and exhaustively dealt with, but nevertheless in a concise way.

Part II is devoted to the elements of the diet. It is subdivided into five chapters. Eggs, milk and milk products, meats, fish and poultry, cereals and sugars, vegetables and fruits, fats, oils and nuts, and miscellaneous foods and condiments are all adequately discussed, and though nothing very new has been introduced in dealing with this question, the subject is handled in a full and thorough manner.

Perhaps Part III, that on dietary treatment of disease, will appeal most of all to the readers of this admirable book. Definite diets and dietary rules are laid down for practically every disease into which the question of food restrictions enters.

Good, practical and most useful diets are given for various deficiency diseases, undernutrition, hyperthyroidism, obesity and overnutrition, gout, chronic arthritis, fever, infections, anemia, nephritis, heart disease and diseases of the circulation, *diabetes mellitus*, acidosis, gastro-intestinal diseases and lead poisoning. A most interesting chapter is devoted to the question of food allergy and its relation to disease, while another practical chapter deals with the most suitable diets in pregnancy and lactation, with special feeding methods, and the diet in surgical conditions, including diabetic surgery. Details are given of the recent work in connexion with the ketogenic diet advocated in cases of epilepsy, which should be of practical value.

In this book the author stresses the very important part that diet may play, not only in causing disease, but particularly the great therapeutic value of a properly balanced and supervised diet of suitable foods in all kinds of diseases. He has put concisely and yet in as detailed a form as necessary, practically all the information necessary for the calculation and prescription of a suitable diet to combat various diseases, information to which easy reference can be made.

This valuable contribution can be confidently recommended, not only to physicians generally, but also to those who profess a special knowledge of this important subject.

¹ "A Treatise on Hygiene and Public Health, with Special Reference to the Tropics," by Birendra Nath Ghosh, F.R.F.P. & S., Revised and Largely Rewritten with the Advice and Assistance of A. D. Stewart, M.B., F.R.C.S.E., D.P.H., D.T.M. & H.; Seventh Edition; 1930. Calcutta: Scientific Publishing Company. Crown 8vo., pp. 754, with illustrations. Price: 10s. 6d. net.

² "Diet in Disease," by George A. Harrop, Junior, M.D.; 1930. Philadelphia: P. Blakiston's Son and Company. Royal 8vo., pp. 412, with eighty tables.

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SCARLET FEVER.

SCARLET FEVER is notifiable in every State of the Commonwealth and in the Federal Capital Territory. In 1929 (the latest period for which figures are available) the number of notifications was 8,249. New South Wales headed the list with 5,229 cases and Victoria was second with 1,490. During 1929 101 persons in Australia died of scarlet fever. When the invalidity likely to result from scarlet fever is considered, and there are no available means of forming an estimate of amount of disability caused, it is evident that scarlet fever still presents an important public health problem. It is perhaps well to recall the fact that statistics of scarlet fever, like those of other infectious diseases, present waves or cycles of increased incidence. Although there are still some who cling to the view that scarlet fever is caused by a filter-passing organism, most workers believe that the *Streptococcus scarlatinae* is responsible. Even those who hold the filter-passing view, admit that the *Streptococcus scarlatinae* plays an important rôle in the production of the clinical picture. It is thus with the *Streptococcus scarlatinae* that most of the bacteriological work in scarlet fever has been carried out.

If attempts are to be made to combat an infective process, the work must be undertaken from two points of view—that of the infecting organism and that of the patient and his bodily condition. There would be no scarlet fever at all, if all infecting organisms could be destroyed, or if susceptible persons could be either placed beyond reach of the infecting organism or so treated that their resistance was raised to a sufficiently high level. The measures at present adopted for the prevention of the spread of the disease are, generally speaking, quite inadequate. Readers are referred to an article on scarlet fever by Dr. A. H. Tebbutt, published in this journal on March 30, 1929. Dr. Tebbutt quoted an interesting extract from a report by Sir George Newman, Chief Medical Officer of the Ministry of Health of Great Britain. Sir George Newman pointed out that, although isolation seems less important and mere disinfection less necessary as time passes, neither can be abandoned because scarlet fever is an infectious disease. On the face of it it seems reasonable to attempt disinfection in the presence of an infective process. Sir George Newman holds that "a vast quantity of popular 'disinfection' which goes on at present, is not only entirely ineffectual, but absurd. To conceal one bad smell by making another is not disinfection." Australian medical practitioners, particularly those in country districts, will endorse this opinion. Disinfection of the patient's home, however, has one advantage in that it may be and often is accompanied by a thorough "spring cleaning." The medical attendant can often suggest this procedure. Cleanliness may or may not be next to godliness, but it is certainly not incompatible with efforts at disinfection.

Since complete reliance cannot be placed in isolation and disinfection, attention naturally turns to the discovery of the causative organism (this includes the question of carriers), the application of the Dick test and the immunization of the patient. It is not to be concluded from this statement that isolation cannot be useful in the checking of an epidemic. This would be absurd. Dr. J. J. Searby reported in these pages on June 26, 1930, the successful isolation of a number of patients in a girls' school. He was singularly fortunate in the

circumstances surrounding the outbreak and he used isolation and bacteriological examinations with good effect. The matter for all that is not so simple as it sounds; the difficulties are many. Limitation of space prevents adequate discussion of the subject and readers are again referred to Dr. Tebbutt's communication. Mention may also be made of certain findings of Gurwitz referred to in Volume VI of the "Annals of the Pickett Thomson Research Laboratory," recently received. Gurwitz based his conclusions on more than 9,000 throat smears taken from 1,083 patients at the Urban Municipal Hospital in Berlin during the previous two years. He found that: (i) Throat smears from scarlet fever patients do not contain haemolytic streptococci in all cases even at the beginning of the disease; in his series they were found in 84% only at the onset of the disease. (ii) During the course of the disease the bacteriological findings vary and do not have any recognizable connexion with the clinical picture. (iii) The bacteriological findings do not bear any relation to the result of the Dick test carried out at the same time. (iv) The absence of haemolytic streptococci is no guarantee that return cases will not occur. Though this is in conflict with work done on the subject in England and America, it is clear that much more remains to be learned about the pathogenesis of scarlet fever. The only method of preventing scarlet fever that offers much prospect of success is the preventive inoculation of susceptible persons by means of vaccines or toxins of the *Streptococcus scarlatinae*. It is not much use expressing a pious hope that medical practitioners will adopt this course. The lead should come from the public health authority. Medical practitioners should be called upon to send in a second report to the department to supplement the notification of the infection. In this report they should be required to specify the steps they have taken to discover carriers and to immunize susceptible persons. If this were done, officers of the department would be able to advise or even to give actual help to the medical practitioner. A great deal has been said about the advisability of cooperation between private medical practitioner and departmental officer. Here is one sphere for cooperation. Whether the cooperation

should take the form suggested may be debatable. It is certain, however, that a radical change must be made; otherwise there can be no advance.

Current Comment.

CLIMATE AND FECUNDITY.

CLIMATE has been blamed for many of the ills of mankind, but it has been proved many times that climate *per se* is a direct factor in the causation of very few diseases. The human organism has a remarkable capacity for adapting itself to its environment; men may live and flourish in the scorching heat of the equatorial zone as well as in the biting cold of the Arctic and Antarctic regions.

Climatic variations call for alterations in the process of metabolism. Of all living things, man seems best able to meet these requirements. An example of the human body's ability to respond to the demands of environment is the increased activity of the blood-forming organs of persons living in the rarefied atmosphere of mountainous districts. This phenomenon is well known. Each or any of the endocrine glands may be called upon to do increased work under some conditions of environment and allowed a greater degree of rest under others; such reactions of the endocrine system are factors in the evolution of racial types. For example, it has been suggested that the Chinese and Japanese, as compared with healthy Western people, are affected with hypothyroidism; in other words, their thyroid glands are not so active as those of the white races. But many factors other than climate may be concerned here. The rather grotesque though fascinating theory has been advanced that in tropical climates there is a depression of the adrenal function, with the result that substances usually eliminated by adrenal action remain in the body and form the pigment which is deposited in the skin of aborigines.

No doubt climatic conditions have an influence on the generative functions in common with the functions of other organs throughout the body. This is exemplified in the increase of the menstrual flow so commonly experienced by women in warm countries. A good deal has been written concerning the effect of residence in the tropics on the generative functions; generally, however, it may be taken that sexual abnormalities are attributable to conditions of existence rather than directly to the climate itself. In this regard an interesting theory has recently been presented by C. A. Mills and Mrs. F. A. Senior.¹ Their investigations revealed that in the very cold northern countries of the American continent the numbers of conceptions are lowest towards the end of winter and highest during the summer weather. On the other hand, in areas in which the mean monthly temperature during part

of the summer is higher than 21.1° C. (70° F.), the conception rate is highest in the winter and lowest during the hottest weather. The higher the rise of summer temperature, the more pronounced is the depression in the rate of conceptions. Mills and Senior note that in most of the districts in which their investigations were made, the highest rates of conception occur when the mean temperature is about 18.3° C. (65° F.) and they remark that this is the temperature which Huntington found to be the optimum for human efficiency. According to their statistics, in Japan occurs the greatest seasonal variation in the rates of conception; the summer rate was 50% lower than the highest rate, which occurred in the spring. Their inquiries led them to conclude that, in this country at any rate, there was increased rather than decreased sexual activity during the warmer weather, and they make the inference that a "biologic reduction in fertility is produced by the heat."

They remark that it cannot be ascertained at present whether only one or both sexes are affected, but they draw attention to the claim of some stock-breeders that a ram loses his powers of procreation during the summer months if his scrotum is kept encased in flannel bandages to retain the heat. They point out that the elevation of the temperature of the testicles by 2° or 3° C. inhibits spermatogenesis.

Occasionally an increase in the marriage rate coincides with an increase in the conception rate, but Mills and Senior declare that the one has little influence on the other and that the definite seasonal variation occurs whether there is a seasonal increase in the numbers of marriages or not.

This suggestion of a seasonal influence on human fertility is an interesting theory. The existence of such a phenomenon is not incredible, but a great deal of further investigation is necessary before it can be accepted as proven. It is scarcely logical to rely entirely on the evidence obtained by the mere plotting of graphs of mean temperature records and other meteorological data together with the numbers of conceptions calculated, as to time, from the date of confinement. It is likely that some factor other than atmospheric temperature may be involved. If Mills and Senior wish to prove their point, it will be necessary for them to undertake a very extensive sociological survey of districts in which their statistics reveal a pronounced seasonal variation and those in which there is no great variation throughout the year. Additional possible factors may be found by means of a study of water and food supplies, racial customs *et cetera*.

That the conception rate is not invariably influenced by a rise in temperature to a monthly mean of 21.1° C. (70° F.) or over is made apparent by a study of the statistics of the City of Brisbane and the metropolitan area of Sydney for the year 1930. In both these districts the monthly birth rate, hence the conception rate, varies little throughout the year. It is perhaps worthy of note

that in Brisbane in 1929 the greatest number of births occurred during April and the least during February; in 1930 the greatest number occurred during October and the least during November. Obviously there is no evidence of seasonal influence here, though it cannot be denied that some such evidence might possibly be revealed by a study of the statistics over a number of years.

While the theory expounded by Mills and Senior cannot logically be accepted without reservation, it is worthy of more than passing thought. Possibly it may be the means of stimulating further research into the effects of climate upon the endocrine glands. Such research is important and necessary, for it may lead to the revelation of valuable information on many, at present, obscure causes of invalidism in the tropics and also to considerable increase in the general knowledge of endocrinology.

THORACOPLASTY IN PULMONARY TUBERCULOSIS.

THERE are three types of operative procedure which may be used in pulmonary tuberculosis—the production of an artificial pneumothorax, phrenic avulsion and thoracoplasty. Comparatively little has been published in this journal in regard to thoracoplasty. It is therefore interesting to note a recent report by B. N. Carter.¹ During a period of five years he has performed fifty-three thoracoplasty operations. Carter points out that the commonly accepted indications for thoracoplasty are a chronic fibrous type of tuberculosis with or without a cavity, essentially unilateral, in a patient who has shown evidence of good resistance, in whom treatment in a sanitarium has failed to effect a cure and in whom artificial pneumothorax cannot be performed. In his opinion the "inflexible indications" should be: (i) evidence of resistance as indicated by fibrous tissue reaction around the lesion, with contraction of the wall of the chest and dislocation of viscera; (ii) the impossibility of performance of a satisfactory pneumothorax. The rapidly progressing caseous types of tuberculosis are not suitable for operation. The average duration of the disease in his patients before operation was three years. Carter's fifty-three patients were subjected to 108 operations—45 to a two-stage and five to a three-stage operation, three patients had only one stage completed. Forty-four patients are alive and nine have died. In three instances death was due to the operation; two of the remainder "should never have been subjected to operation." Limitations of space preclude further description of Carter's work. His conclusion will possibly evoke criticism, but his records show that he is justified; he states that "in a case suitable for collapse therapy, but in which this may not be thoroughly accomplished by pneumothorax, it is far safer to do a thoracoplasty, and this should be done early and not as a last resort."

Abstracts from Current Medical Literature.

RADIOLOGY.

Cholecystography.

SHERWOOD MOORE (*The Journal of the American Medical Association*, December 27, 1930) gives an analysis of the results of cholecystography. The intravenous method of administration is used as a routine, the oral method being applied only in certain cases. The method is a measure of hepatic function as well as gall bladder function. Of the total patients 66% gave no reaction to the drug, 19.7% gave a mild reaction and 14.3% a second degree reaction and none a third degree reaction. Cholecystographic estimation of the condition of the gall bladder is based on the fact that the normal organ receives the dye, concentrates it and alters in size, finally evacuating its contents. A diseased gall bladder has its functions interfered with in a manner proportionate to the degree of impairment of the organ. There have been instances in which the radiologist considered the cholecystographic behaviour normal, though the symptoms were those of cholecystitis. In all these cases there were extensive adhesions and pericholecystitis. The peri-cholecystitis originates in two ways: (i) From the gall bladder itself and in these circumstances there is total absence of concentrating function, or (ii) from extrinsic sources, when the gall bladder function is apparently normal, with rarely demonstrable deformity or fixation. Calcified gall stones constituted 59.1% of the total stones diagnosed, the remaining 40.9% being diagnosed as "negative shadows." Low in the scale of accuracy of pre-operative diagnosis is the cholecystogram which reveals a "thin" or "faint" shadow and such findings need wary interpretation.

Intestinal Tuberculosis.

C. WU AND C. K. HSIEH (*National Medical Journal of China*, October, 1930) give a Röntgenological study of intestinal tuberculosis. After discussing the pathological anatomy and technique of the examination, both by barium meal and barium enema, the authors give a description of the normal intestines and a table of the times at which the various portions can be visualized. The dependable cardinal signs of intestinal tuberculosis in order of importance are: Filling defect, colonic hypermotility, ileal stasis and dilatation. The filling defect in the ulcerative type is the result of local irritative spasm and in the hyperplastic type of encroachment on the intestinal lumen a hyperplastic lesion may prevent the flow of a barium enema into the portion proximal to the obstruction. An ulcerative lesion produces an irregularity of the intestinal wall which may change in contour from time to time. Hyperplastic masses narrow or constrict the lumen in permanent patterns. A filling

defect must be differentiated from muscular contraction, the presence of faecal matter, gas, transitory spasm, adhesions or extrinsic pressure. Colonic hypermotility is seen by an acceleration of the flow of the barium meal into the colon, a phenomenon observed only in the presence of an ulcerative lesion, particularly an irritative lesion of the ileo-caecal intestine. Ileal stasis is the retention of all or part of the barium meal on the ileal side for periods longer than is normal. It is the result of obstruction by the presence of a hyperplastic mass or the intermittent spasm produced by an ulcerative colitis. In the ulcerative type it is generally milder and is associated with colonic hypermotility. In hyperplastic lesions the obstruction is mechanical and dilatation of the terminal part of the ileum is present. The authors establish a differential diagnosis from chronic appendicitis, carcinoma, non-tuberculous chronic colitis and parasitic diseases.

Effusion of the Thorax.

E. FREEDMAN (*Radiology*, January, 1931) writes on the X ray appearance of interlobar and mediastinal encapsulated effusion in the thorax. Interlobar effusions are characterized by sharply defined band-shaped or wedge-shaped or circular shadows in the region of the interlobar septa. Lobar and marginal pneumonia, localized and circumscribed caseous consolidations and bronchial carcinomatous must be differentiated from these effusions. In the presence of pneumonia the only radiological sign of an interlobar effusion is the bulging of the interlobar fissure seen in the lateral view. Mediastinal pleural effusions are represented by band-shaped, wedge-shaped or triangular shadows parallel to the vertebral column or to the cardiac silhouette. Pericardial effusions, paravertebral abscesses, aortic aneurysms, mediastinal tumours and areas of bronchiectatic consolidation may give a similar radiological appearance. Bronchography, in the author's opinion, is a valuable aid in the diagnosis of both interlobar and mediastinal encapsulated effusions.

Dwarfism and Disordered Epiphyses.

HOWARD E. RUGGLES (*American Journal of Roentgenology*, January, 1931) describes dwarfism due to disordered epiphyseal development. The condition appears to be a familial disease. The children are short, the head is large, the eyes widely separated, the root of the nose depressed, the sternum narrow and prominent with kyphosis in the thoracic or lumbar regions. The hips are somewhat flexed, there is pronounced *genu valgum*; there is loss of power in the arms and legs, but no muscular atrophy. The intelligence is unimpaired. The laboratory findings are normal; the Röntgen findings are characteristic. The process is a profound disturbance of epiphyseal development. The calvarium is normal. The *sella turcica* may be small with heavy clinoid processes. The ribs are heavy. The bases of the metacarpals

are pointed, ossification begins in the distal row of carpal bones and they are all small and irregular in outline. The heads of the femora are usually absent, the acetabula are eroded upwards and irregular in outline. In the knees there is definite loss of substance of the outer condyles of the tibiae and an overgrowth of the inner condyles of the femora. There is a relative shortening of the fibulae. The tarsal bones are extremely irregular and the bases of the metatarsals pointed. The vertebral bodies are wide and flat and very irregular, and the intervertebral discs are as thick as the bodies. Achondroplasia is the disease most resembling this condition, but the radiological, and especially the physical, characteristics of the children differ.

PHYSICAL THERAPY.

Light Therapy in Lupus.

RICHARD VOLK (*British Journal of Actinotherapy and Physiotherapy*, October, 1930) states that a choice of apparatus for the light treatment of lupus lies between the Finsen-Reyen and the Kromayer lamps. The former is designed on the same principle as the large Finsen lamp, but for a less powerful current and for treating one patient at a time. Comparisons between the clinical effects of the Finsen and Kromayer lamps prove that the deep seated effect of the latter is less than that of the former. The lesser penetration is due principally to the nature of the light, which produces a strong superficial reaction. Attempts have been made to remedy this by inserting a blue quartz disc to filter off the ultra-violet rays of very short wave lengths. Whatever the importance in lupus treatment of local phototherapy which acts on diseased foci only, general phototherapy should not be omitted. A combination of the two has become standardized in almost all institutions where lupus is treated. Volk considers the proper proportions of actinic and heat rays to be of paramount importance; owing to their greater penetration, the heat rays may possibly heighten the effect of the ultra-violet light. Exposures to carbon light are governed by the same general rules as treatment by sunlight. Regarding duration of exposure, the author keeps a mean between Rollier's graduated dosage method and Reyn's method of producing at once a strong erythema by about half an hour's exposure and adding ten to fifteen minutes every other day, leading up to a daily exposure of two to two and a half hours without any interval for depigmentation. All cases of *lupus vulgaris* are suitable for treatment by carbon arc light, their great amount of infiltration and extent not permitting efficient treatment with the Finsen or Kromayer lamp; the same applies to ulcerated and hypertrophic forms, the focus of which is prepared for Finsen therapy by previous X ray treatment. Apart from general irradiation, the affected part is sub-

jected to intensive treatment by placing it as close as possible to the source of light, fixing it in position by special apparatus when necessary and applying preliminary cautery to increase the local effect. Very good results have been obtained with papulonecrotic tuberculosis and indurative tuberculosis. Surgical tuberculosis of bones, joints and lymphatic glands reacts very favourably to carbon arc light, particularly when this is combined with X ray treatment. The "artificial alpine sun" lamp ought not to be applied by routine. Starting with exposures of five minutes at eighty to one hundred centimetres distance, the dose may be increased by lengthening the time and reducing the distance. Care should be taken that all parts of the body are about the same distance from the light, so as to avoid too strong reactions or burns.

Röntgen Therapy in Goitre.

GEORGE E. PFAHLER AND JACOB H. VASTINE (*American Journal of Roentgenology and Radium Therapy*, October, 1930) report that simple colloid or non-toxic goitres are treated by them only when they have failed to yield to medical treatment or when operation has been refused. Hyperplastic goitre is a non-toxic disease of adolescence. Only three patients have been treated, but in all the enlargement of the gland disappeared. One of these patients passed through pregnancy later without a recurrence. Of the patients with toxic hypoplastic or exophthalmic goitre 84% were females. The methods of treatment available are medical, radiological or surgical. In their opinion a combination of the two former methods is the procedure to be adopted in most instances. Medical treatment of non-toxic adenoma is ineffectual in giving relief or retarding the progress of the condition. Iodine is definitely contraindicated because it not infrequently converts an innocuous adenoma into a severe toxic one. Small localized adenomas are those in which irradiation offers the most hope of improvement. Large adenomas are better treated surgically than radiologically. In a series of 92 patients with toxic adenoma 91.8% were either cured or definitely improved. None of the patients developed symptoms of hypothyroidism following irradiation and in none was there manifested any evidence of other unpleasant sequelae except telangiectasis. Surgical removal of substernal goitre is difficult and attended by a rather high mortality. The possibility of mediastinitis is always great following surgical interference. Twelve patients with substernal goitre were treated; six of them had exophthalmos and their goitres could be regarded as of that type. A disappearance of the goitre was effected in five of these cases. There were 23 cases of goitre in which thymic enlargement could be definitely recognized by Röntgenological means. Twenty of the patients are stated to be cured and two greatly improved by

irradiation of the anterior mediastinum and thyroid region. In regard to technique, the anterior cervical region is divided into four areas, two anterior and two lateral. The larynx is protected with lead. The rays are directed medially and downward so that a cross-firing effect is produced. Frequent metabolic determinations should be made. Routine dental examinations are made to determine the presence of focal infection. A routine chest examination is made to record the size of the heart, determine the presence of thymic enlargement or substernal goitre. Associated medical treatment is a very important consideration in the management of goitre. It is the authors' opinion that the use of iodine in conjunction with Röntgen therapy is not generally wise. Iodine should not be given to patients with toxic adenoma and only in selected cases of exophthalmic goitre. The general medical care of the patients is left to the discretion of the physician.

Radiation and Cancer of the Uterine Cervix.

F. A. TYLOR (*Archives of Physical Therapy, X Ray, Radium*, December, 1930) states that radiation treatment has been employed so long in the treatment of cancer of the uterine cervix that technique is well standardized and the pathology understood. Statistics are available showing what can be accomplished by this method of treatment. It is advantageous to divide cancers of the uterine cervix into certain groups. The division advocated by Burnam has proved valuable to the author: (i) Small cancers confined entirely to the cervix; (ii) cancers of the cervix which have extended on to the vaginal wall; (iii) cancers of the cervix which have extended into the parametria (blood, ligaments, vesico-vaginal septum or recto-vaginal septum); (iv) cancers of the cervix which have metastasized to distant parts (periliac or periaortic glands, bone *et cetera*). The greatest contraindication to radiation is the presence of pelvic infection. There has been no question at any time since radiation therapy was introduced about inoperable cancers. Patients with this class of growth have always been helped by X ray or radium or by a combination of the two. It took more time, however, to prove the value of radiation in the treatment of operable growths. The use of radium alone has been well established by the users of radium as most suitable for Burnam's class (i) and class (ii) cancers of the cervix. The rectal mucosa is exceedingly sensitive to radiation, much less than the "carcinoma dose" causing ulceration and sloughing. Neglect to protect the rectum causes much post-irradiation suffering and even may produce recto-vaginal fistula. The author considers that the position recommended several years ago by H. H. Bowing is the only satisfactory one in the application of radium to the cervix. He puts the patient in the knee-chest

position. As soon as the speculum is inserted, air fills the vagina, the abdominal viscera fall down and a perfect view of the central vaginal canal and cervix is possible, with all folds obliterated and the cervix in the centre of the vault. The radium applicator can be slipped into the cervical canal and the packing done with the greatest ease. Only in the exceptional case should the dorsal position be used. In Burnam's class (iii) and class (iv) where the cancer has invaded the parametrium and lymphatic glands around the iliac arteries of the aorta, high voltage X radiation is the method of choice.

Pruritus and Eczema of Vulva and Anus.

AGNES SAVILL (*The British Journal of Actinotherapy and Physiotherapy*, December, 1930) states that the treatment of pruritus and eczema of the anus is often described together with that of the vulva because the entire ano-genital region may be affected by the inflammatory cutaneous reaction to the exciting cause. Before a plan of treatment is determined the cause must be investigated and, if possible, removed. A discharge from cervix or vagina is the most frequent cause of vulvar pruritus and eczema. Other frequent causes are cystitis, urethritis and urinary infections from various sources; the latter must be borne in mind as an often unsuspected cause. Constipation, colitis, piles and fissures are frequent causes of anal pruritus and eczema. The exudation of mucus, leaking paraffin or an attack of diarrhoea may start pruritus in an area which, when scratched or rubbed, develops quickly into an angry eczema. The presence of worms must always be investigated; even in adults thread worms may be present. During recent years a mycotic infection has become common; this may attack only one small part or may extend over a wide area, including the anus, intergluteal fold, vulva, groins, even abdomen and thighs. In the early stages it is recognized by the fine branny scales with circinate margin; with the microscope or on culture the diagnosis is made certain. In the management of every case there are two points of paramount importance. First and foremost, all scratching must be forbidden; swelling and destruction of the new forming cells must not be brought about by scratching. The second important point in local treatment is the prevention of contamination of the irritated part. This is guarded against by applying vaseline or oil before passage of stools or urine, by careful cleansing after and by keeping the parts dry. X rays are the most potent of all therapeutic agents. X radiation was the method of election from about 1905 till after the war. In 1904 the author made a special investigation into the question of X ray dosage in the treatment of lupus and other skin diseases. It is impossible to give definite directions as to X ray dosage, because every apparatus differs.

Special Articles on Diagnosis.

(Contributed by Request.)

XL. HEAD INJURIES.

THE immediate diagnosis of head injuries is always uncertain, because the pathological interpretation of the clinical phenomena is at most doubtful, often fallacious and sometimes impossible. "No head injury," wrote Hippocrates, "is so severe that it should be despised of or so slight that it may be neglected."

Clinical observation is the keystone of diagnosis and receives but little support from X rays and other methods. Whilst it is true that X rays give valuable information of fractures of the skull, in civil practice these play no great part. It is the assessment of the brain injury, direct or indirect, that is the all-important aim of diagnosis.

Many structures may be damaged by the injury—the scalp, the skull, the meninges, the meningeal arteries, the venous sinuses or the brain. The ideal of diagnosis would be to identify the exact nature, site and extent of injury to each of these separately. In scalp and skull injuries this can usually be done, but in the identification of intracranial injuries, however, considerable difficulty and doubt may arise, for the reason that injury to the meninges, meningeal arteries, venous sinuses *et cetera* usually manifests itself through the single medium of the brain, which is also injured. As an example, hemorrhage from a meningeal vessel gives no signs directly referable to the vessel or to hemorrhage as such—pallor, thirst, falling blood pressure and rising pulse are all absent—instead, it is the effect on the brain of the blood within the cranial cavity that gives rise to the clinical picture. Thus in any head injury one must consider its effect upon: (i) The scalp, (ii) the skull, (iii) the intracranial contents.

INJURY TO THE SCALP.

Wounds of the scalp usually appear to be incised, even though caused by blows or blunt objects. When they extend through and across the aponeurotic layer, they gape widely. They bleed freely and are frequently contused. All scalp wounds should be examined with great care for evidence of fissured or depressed fracture. In all punctured wounds of the scalp depressed fracture should be especially excluded. Contusions are very tender.

Hæmatomata of the subcutaneous tissues have soft centres and firm edges. Their extraordinary simulation of depressed fracture would be disconcerting were it not for the fact that depressed fractures are almost invariably compound, except in the new-born and very young. The margins of a hæmatoma are raised above the surface of the skull and can be dispersed by firm pressure. X rays will give finality to any doubt.

Hæmorrhage beneath the subaponeurotic layer may be localized or limited only by its attachments. When localized, it is not very obvious. The scalp pits on pressure over it. In recent cases this pitting should be diagnosed as subaponeurotic hæmorrhage with a provisional diagnosis of fissured fracture of the skull.

Tenderness and hæmatomata in the temporal region should always arouse a suspicion of middle meningeal hemorrhage. At times large and rapidly increasing hæmatomata are seen in the temporal region in patients, particularly children, with severe general symptoms.

Although injury to the brain and intracranial contents does not necessarily lie beneath the point struck, it is usual for sharply localized injuries to cause localized trauma. Diffuse blows give rise to widespread and even remote injury. *Contrecoup* injury is common and blows on the back of the head, as a rule, affect mainly the tips of the temporal lobes and the under surface of the frontal lobes.

FRACTURE OF THE SKULL.

Fracture of the skull is found in about one among every ten patients with head injury admitted to hospital. As there is no alteration in the function of the bone; unless the fracture is compound, the clinical evidence of fracture is the result only of disturbance or injury to contiguous structures. The severity of the general signs of head injury gives no definite clue to the presence of a fracture, which may be found in patients who have merely been stunned, or may be absent in patients who are comatose. Unless X ray examination is carried out as a routine in all head injuries many fractures will fail to be diagnosed.

For clinical purposes these fractures are divided into two main subdivisions: fractures of the vault and fractures of the base.

Fractures of the Vault.

Fractures of the vault may be simple or compound, fissured or depressed.

Simple Fissured Fracture of the Vault.

Simple fissured fracture is definitely diagnosed only by X rays. Subcutaneous emphysema of the scalp, though rarely seen, indicates a fracture involving the frontal sinus or mastoid air cells. The presence of a fissured fracture may be presumed when the scalp pits on pressure over a subcutaneous hæmatoma. When fracture is present, particularly in children, the skull may give a cracked pot sound on percussion. In the X ray diagnosis arterial grooves and diploic channels frequently cause errors of diagnosis.

These fissured fractures do not necessarily affect the immediate or remote prognosis of the head injury, although it is difficult to convince the public in general, and juries in particular, that this is so. They require no special treatment and in the absence of cerebral symptoms trephining is unjustifiable.

Compound Fissured Fracture of the Vault.

Compound fissured fracture is diagnosed by seeing the fracture in the depth of the wound. It appears as a wavy, irregular line across the skull, usually oozing blood. When this is seen, there is very little doubt about the diagnosis, although sutures may lead to error. Examination of the wound with a probe is misleading, as not only sutures, but also torn edges of fascia may simulate fractures when thus examined. Brain matter oozing from a wound is, of course, definite evidence of fracture and torn meninges.

Depressed Fracture of the Vault.

Depressed fracture is typically compound and the diagnosis from hæmatomata of the scalp has already been mentioned. The injury to the inner table is greater than that to the outer, and the depressed piece of bone is usually comminuted. The diagnosis is made by examination of the wound or by X ray examination. It is sometimes indicated in the frontal region by a large obvious dent. When these fractures occur in the middle line, they often injure the longitudinal sinus. The bleeding, which is usually not very free until the depressed pieces of bone are elevated, is easily controlled.

In young children simple depressed or "pond" fracture is found, particularly in forces injury to the new-born child.

Fractures of the Base.

Fractures of the base usually result from severe injury and are often associated with signs of extreme intracranial injury, although not necessarily so. The injury which causes them is received on the vault or the sides of the skull and then the fracture runs by fissuring to the base. The diagnosis by X rays is not so reliable as in fractures of the vault. The evidence is essentially clinical and the diagnosis is made on signs such as the escape of cranial contents (brain matter and cerebro-spinal fluid) from the nose or ear *et cetera*, extracranial hæmorrhage and injury to the various cranial nerves. These signs vary with the site, that is, whether in the anterior, middle or posterior fossa.

Fractures of the Anterior Fossa.

In fractures of the anterior fossa a definite diagnosis is made by an escape of brain matter or cerebro-spinal fluid from the nose or by X ray examination. Escape of air into the subcutaneous tissues (surgical emphysema) or into the cranial cavity (intracranial pneumatocele) is rare, but when present denotes a fracture involving the frontal sinuses. The escape of cerebro-spinal fluid from the nose (cerebral rhinorrhea) is definite evidence of fracture of the cribriform plate, but its recognition is obscured by bleeding. Later, a clear watery discharge, which is increased by tipping the head forward, may occur. Orbital haemorrhage is a presumptive sign of fracture of the anterior fossa. This must be distinguished from haemorrhage into the eyelids which causes the ordinary "black eye," frequently present in head injuries. Large orbital haemorrhages may cause protrusion of the eye, though this is uncommon. The late appearance of evidence of orbital haemorrhage has special significance in the presumptive diagnosis of fracture of the anterior fossa, particularly when the injury has not occurred in the region of the eyes. The blood accumulates at the bottom of the orbit, causing sometimes a fullness and discolouration of the lower lid; oedema of the conjunctiva may occur or the whole of the conjunctiva may be lifted from the sclera by the bleeding giving the appearance known as "red eye." The appearance which is particularly regarded as being due to fracture of the anterior fossa, is a fan-shaped bruising appearing on the lateral aspect of the eye, beneath the conjunctiva, several days after the injury. It can be differentiated from purely conjunctival haemorrhage by the fact that it lies behind the conjunctiva, the posterior limit cannot be seen and it cannot be moved with the conjunctiva.

Fractures of the Middle Fossa.

Fractures of the middle fossa are particularly associated with severe intracranial injuries, but not always. Patients are occasionally seen with definite evidence of fracture in this region and with symptoms of only slight concussion. Such examples are by no means infrequent. Definite evidence of fracture of the middle fossa is the escape of brain matter or cerebro-spinal fluid from the ear. Whilst the former is rare, the latter is occasionally seen. At first admixed with blood, later the cerebro-spinal fluid may be recognized by its clear, limpid, watery character, so aptly designated "*eau de cristal*" by the French.

Of the cranial nerves, the facial nerve is most commonly injured. The facial paralysis may appear immediately or not for several days and is not necessarily permanent. This may be associated with deafness, due to injury to the auditory nerve, although this deafness may be due to direct injury to the cochlea *et cetera* by the fracture. The trigeminal nerve is rarely injured.

Haemorrhage from the external auditory meatus, not due to local injury, is in itself sufficient for a clinical diagnosis of fracture of this fossa. In such cases the ear drum is always ruptured. The bleeding may be slight, intermittent or severe. When copious in an unconscious patient, it may be a valuable localizing sign of middle meningeal haemorrhage. Sometimes no bleeding may occur, but on otoscopic examination blood may be seen pent back by an unruptured ear drum.

A diagnosis of fractured base is unwarranted, however severe the injury, unless one or more of the above signs are present.

Fracture of the Posterior Fossa.

The only definite means of diagnosing fracture of the posterior fossa is by X rays. Bruising near the tip of the mastoid or appearing later in the posterior triangle is suggestive. Paralysis of the hypoglossal nerve may occur, but is very rare.

INJURY TO THE INTRACRANIAL CONTENTS.

When injury to intracranial contents occurs there are three main factors which may determine the clinical manifestations. The first is a general brain injury which gives rise to the clinical picture known as "concussion." It is associated with very little, if any, gross change in the brain. The exact pathology of the condition is indefinite

and is the subject of many theories (Trotter, Duret *et cetera*).

The second factor is cerebral oedema, which may be primary or secondary to actual contusion or laceration of the brain substance; its clinical features are expressed in the term "cerebral irritation."

The third factor is an acute increase in the intracranial tension, the symptom complex of which is known as "compression." Haemorrhage particularly is the causal agent.

As there is but one medium of expression of symptoms, the brain, in each of these intracranial changes, it is not surprising that at times the diagnosis of the exact pathological lesion or lesions—for they are often multiple and bilateral—is difficult and frequently impossible. The difficulty is not lessened by the fact that an injured brain may be eccentric and fantastic in its reactions.

Thus the general signs and symptoms are the result of irritation or paralysis of parts or the whole of the brain and the diagnosis of the exact pathological lesion depends on a consideration of the three factors mentioned above together with the clinical evidence of localizing or other signs of brain injury. As a result of this, certain clinical syndromes have come to be recognized as denoting certain pathological lesions.

The following clinical phenomena are recognized as having particular value in the diagnosis of head injury.

1. *The Pupils:* Variation in the size of the pupils is common. The pupil on the side of a compressing factor, as pointed out by Sir Jonathan Hutchinson, first contracts and then becomes dilated and fixed. The opposite pupil, at first normal, later goes through the same changes, thus at first causing inequality of the two pupils. This was said to occur in 25% of cases. Whilst it is true that a fixed and dilated pupil is a localizing sign of value, nevertheless the experience of many observers has shown that inequality of the pupils alone has not the full significance originally attributed to it. Unequal but active pupils are only of value when considered in conjunction with other signs, and are certainly not evidence *per se* of middle meningeal haemorrhage. They may be seen in simple concussion. Whilst they should be regarded as signals of possible danger and the patient should be carefully watched for signs of compression, this sign alone is not an indication for operation. A glass eye has been known to cause considerable anxiety in diagnosis.

2. *Temperature:* Phelps has shown that the temperature gives a most important guide to prognosis and that those patients found to have a temperature over 38.9° C. (102° F.) on admission, rarely, if ever, recover. Most patients on admission have a subnormal temperature which rises during and after the stage of reaction. Those who recover (excluding those with fever from other causes), usually do not develop a temperature over 38.3° C. (101° F.). With temperatures over 38.9° C. (102° F.) the prognosis is usually serious and becomes the more alarming the higher the temperature.

3. *Focal Signs:* Twitching of muscles, paralyses, aphasia, Jacksonian epilepsy or general convulsions may occur. Epileptic phenomena, whilst of localizing value, are not necessarily signs of increasing compression, but may occur in simple concussion, which passes to an uneventful recovery. Paralyses are of great localizing value and are usually due to haemorrhage. Increasing paralysis is evidence of increasing compression. In unilateral paralysis the lesion should be diagnosed on the side opposite the paralysis, although this is not always so. Paralysis of the facial muscles and deafness in one ear, and paralysis of cranial nerves in general are more likely to be due to a fracture of the base of the skull than to a localized compression. With middle meningeal haemorrhage there may be found any variety or the absence of any paralysis.

4. *Lumbar Puncture:* Apart from its therapeutic value, lumbar puncture may give important diagnostic evidence. Blood-stained cerebro-spinal fluid is diagnostic of intradural haemorrhage, though clear fluid does not necessarily exclude it. It is unwise to draw conclusions from the apparent tension of the cerebro-spinal fluid, particularly on a second or later puncture.

5. **Pulse and Blood Pressure:** Conclusions drawn from the pulse rate are of value only when considered in conjunction with the state of the patient's consciousness. It is extremely common for patients with concussion to have a very slow pulse rate, which persists even for weeks during the recovery period. In a conscious patient this slow pulse has no special significance. In an unconscious or semi-conscious patient slowing of the pulse rate is evidence of increasing compression and is an unfavourable sign. All patients admitted with a head injury should be put on an hourly pulse chart for at least twelve hours after admission and this should be continued so long as the patient is not conscious. In increasing compression this fall in the pulse rate is associated with an increase in blood pressure and it is during this period that any operation for the relief of the condition should be performed. If the operation be delayed too long the pulse rate will begin to rise and the blood pressure to fall. The patient is then moribund. Except in children, in whom the pulse rate is usually higher than adults, pulse rates over 120 are signs of extremely ill omen.

6. **Ophthalmic Signs:** Ophthalmoscopic examination cannot be relied upon to give much information of value, either of compression or its localization.

7. **X Ray Examination:** X ray examination gives definite evidence of the presence of fissured and depressed fractures of the vault. Fractures of the anterior and posterior fossæ can as a rule be definitely diagnosed, but in fractures of the middle fossa X ray diagnosis is not reliable. An X ray diagnosis of a fracture in one temporal region may be of some localizing value in middle meningeal haemorrhage, but these patients are usually too ill to be subjected to the examination.

8. **Changes in Consciousness:** Changes in consciousness are particularly indicative of trauma to the intracranial contents. These changes may vary from slight stunning or dizziness to complete coma. Any change in consciousness is diagnostic *per se* of some damage to the brain, though the converse is not true. Whilst the condition of consciousness of a patient on admission soon after a head injury is an important guide to prognosis, the most valuable information is obtained from the progress or retrogressions of consciousness which may take place. These changes in consciousness offer the most satisfactory basis for the clinical grouping of these cases.

The recognition of the various degrees of consciousness is of the greatest importance and they are familiar to all trained in the administration of anaesthetics. This knowledge of the changes in consciousness during anaesthesia can be applied to the diagnosis of head injuries with advantage.

In anaesthesia consciousness is lost in the following order: Full consciousness, dulled consciousness, semi-consciousness, light unconsciousness, deep unconsciousness, coma, and regained in the opposite order. After a head injury the consciousness of the patient can usually without much difficulty be placed in one of these stages. During recovery he passes through the stages to full consciousness. The process of recovery may become arrested or after a complete or partial recovery may retrogress. The recognition of these stages gives important clues to the changes which are taking place within the cranial cavity, and they are important guides in the decision to operate, which is one of the most difficult and anxious decisions in the management of head injuries. At the Saint Vincent's Hospital attempts have been made to chart these changes in consciousness with some success, but the difficulty of making such charts is great. The use of sedative drugs, required at times to quieten restless patients, is an added difficulty to the proper assessment of these changes in consciousness.

Head Injuries and the State of Consciousness.

Analysed from the point of view of changes in consciousness, head injuries fall into four main groups:

1. Those patients in whom there is a disturbance of consciousness varying from an immediate and transient loss of consciousness (that is, a slight stunning) to semi-consciousness lasting for some hours, and who then recover and maintain full consciousness. Of patients admitted in this state 95% recover.

2. Those patients admitted unconscious or deeply comatose. Of these many die within two hours and 60% are dead within twelve. The intracranial damage in such cases is extensive and usually associated with severe haemorrhage.

3. Those patients who are admitted in a semi-conscious state, who do not recover full consciousness, but who may remain drowsy and delirious for days or pass into the state recognized as cerebral irritation. Such a train of symptoms is due to cerebral oedema or certain types of subdural haemorrhage.

4. Those patients who recover consciousness to some extent and then progressively relapse. There are several varieties: (a) Those who recover consciousness completely and then relapse into unconsciousness. This is known as the "latent interval" type and is typical of extradural haemorrhage. (b) Those who only partly regain consciousness and then relapse. This may be due to extradural haemorrhage, but is particularly associated with intradural bleeding. (c) Other latent interval types, to be described later, due to cessation and recurrence of bleeding from cerebral veins or the bursting of cerebral hemorrhages into the subarachnoid space or into the ventricles.

It is now necessary to examine in detail the symptom complex of concussion, cerebral irritation and compression.

Concussion.

Concussion characterizes the majority of head injuries and the progress is very typical. Except when the injury is almost immediately fatal or when it is complicated by haemorrhage, laceration *et cetera*, almost all the patients recover. In the early stages it is impossible to foretell what complications may ensue.

Usually on receipt of the injury the patient completely loses consciousness and collapses. The loss of consciousness may be merely momentary, may be merely a slight stunning or a state of semi-consciousness lasting for several hours or more. Following this the patient may immediately regain consciousness and suffer from headaches and dizziness of varying duration and intensity, but he often remains dazed or semi-conscious for several hours, when he recovers consciousness somewhat rapidly. During this time he exhibits all the signs and symptoms of shock. Twitching of the face or limbs or even fits may occur, the pupils are usually equal, but may be unequal. The patient throughout is semi-conscious and not fully unconscious. After a while the stage of reaction occurs and he recovers consciousness. He stirs and moves his limbs, his pulse becomes stronger, his blood pressure rises and his colour improves. Vomiting is the particular feature of this stage and definitely demarcates it. In a normal case return to consciousness occurs at this stage and is maintained. Should the patient not regain full consciousness or again become unconscious, then the condition is one either of cerebral irritation or of compression. In all head injuries this stage at which vomiting occurs is the crisis and the progress of patients during and after this stage is of the greatest prognostic importance.

In concussion the patient is never less than semi-conscious and at most never beyond that stage recognized by the anaesthetist as "just going under." Almost invariably he shows some signs of consciousness, either by groaning, by resisting attempts to open his eyelids, by rolling his eyes, moving his limbs or resenting interference—a conjunctival reflex is always present—on admission these patients are never completely unconscious or stuporous. Return to consciousness is somewhat rapid and is maintained with perhaps slight drowsiness at first. It is usual for the patient's mind to be a complete blank concerning events prior to the stage of reaction or even for some time prior to the accident. Automatism during this period is common. Football provides the commonest examples of this, where, frequently following a kick on the head, players play throughout the rest of the match automatically and can later recollect nothing that has occurred. It is the rule for children to be extremely drowsy, but usually after a good sleep they wake up quite well. In the convalescent period headache and giddiness are frequent complaints. The pulse rate is often surprisingly slow and rates below 60 are common and may persist

for weeks. As mentioned above, a slow pulse rate in a conscious patient is not a sign of compression or any indication for operative interference.

Cerebral Irritation.

Cerebral irritation is never a primary condition and either follows preliminary concussion or occurs during recovery from compression. When it is preceded by the symptoms of concussion it is usual for it to appear either immediately after the stage of reaction or after an interval of drowsiness or partial consciousness. Throughout, the patient is dazed and drowsy and his condition is typical and easily recognized. He lies curled up on one side in bed, with all limbs flexed, he has an intense photophobia, and his eyes are tightly closed; he is extremely irritable in temper and restless. Usually these patients remain fairly quiet, unless disturbed, when they exhibit considerable irritability of temper. Faeces and urine are frequently passed involuntarily into the bed. The symptoms may persist for a week or more, when consciousness gradually returns. During the period of recovery these patients are often childish and fatuous, though these mental symptoms rarely persist.

Some patients are extremely noisy and restless, requiring restraint by a restriction sheet, and are delirious, especially at night. In these cases prognosis is bad and the symptoms are probably associated with slow haemorrhage or spreading cerebral oedema.

Other variations occur, in which the patients, although they do not present the typical features of "cerebral irritation," remain drowsy and torpid for hours or days after the period of reaction.

Compression.

Death as a result of head injury generally, except in immediately fatal cases, is due to compression. Its recognition in the early stages is often difficult. Nevertheless early recognition is essential, because if treatment is delayed until signs of severe bulbar compression appear, nothing will be of any avail.

Kocher divides the signs and symptoms of compression into four stages:

1. Stage of compensation, in which diminution of the cranial cavity from 5% to 6% may be tolerated with few or no symptoms beyond slight headache, mental dulness and focal signs depending upon the site of the compressing factor.

2. Initial stage of compression, in which headache and mental dulness, excitement, delirium and restlessness occur with diminishing pulse rate and a slight rise in blood pressure and temperature.

3. The stage of fully developed compression, in which the patient becomes unconscious with slow, bounding pulse and raised blood pressure.

4. The paralytic stage, in which the patient is deeply unconscious, his muscles are relaxed and pupils dilate widely, the pulse rate rises and the blood pressure falls. Respiration is shallow and irregular and all the medullary centres gradually fail.

Haemorrhage is the most common cause of compression. The bleeding may arise from the meningeal arteries, the venous sinuses, the internal carotid or cerebral arteries or the cerebral veins. Meningeal haemorrhage is as a rule from the middle meningeal artery and is usually extradural, except in children, in whom extradural hematoma is rare. Haemorrhage from the internal carotid artery is intradural and as a rule rapidly fatal. Bleeding from venous sinuses may pass externally through a fissured fracture or be subdural. From cerebral veins the bleeding is subdural.

Bleeding may thus be extradural, subdural or cerebral. When subdural, it may be diffuse, localized or latent. The cerebral type has an especial tendency to burst after a long latent interval either into the subarachnoid or subdural space or into the ventricles.

If the haemorrhage has a free outlet through a compound fracture, no symptoms of compression may occur. In such cases it is not uncommon for the manifestations of head injury to be but slight or for the patient to be actually

walking about with blood and brain matter oozing from the wound. In rare instances the blood has a partial outlet. This is seen particularly in children. The blood seeps through a linear fracture and collects in the sub-aponeurotic layer of the scalp or in the temporal fossa. At this stage the signs of intracranial injury are not usually severe. The haematoma can be seen to increase until a stage is reached, when acute signs of compression appear. This is sometimes described as the "safety valve haematoma." When there is no outlet for the blood, the bleeding directly encroaches on the closed space of the cranial cavity. Except when rapidly fatal, such bleeding is usually not continuous in its effects, but shows variations due to alterations in blood pressure, cessation and recurrence of bleeding or the bursting of haematomata.

It will thus be seen that bleeding within the cranial cavity will vary in its effects according to whether the skull is closed, semi-closed or open, and further that intervals of comparative quiescence may occur. These remissions give most important clues to diagnosis.

Several clinical types of compression occur:

1. The first type comprises those patients who on admission are deeply unconscious or comatose, in whom the unconsciousness corresponds to at least deep anaesthesia. On examination they are obviously seriously injured and shocked. Both eyes are usually blackened and swollen, signs of fractured base are often present, their limbs are completely flaccid and their breathing stertorous. Other serious injuries are usually present. The temperature is usually subnormal, but may be high, the pulse may be slow or rapid. A high temperature or rapid pulse is particularly unfavourable. Those with a temperature over 38.9° C. (102° F.) rarely recover, whilst those with hyperpyrexia or rapid pulse and Cheyne's Stokes breathing invariably die. The majority of such patients die within two hours of admission. In all, 66% die in spite of any treatment. These patients are frequently the subjects of injudicious immediate surgical interference which usually results in death on the table. The lesions in these patients are usually extensive brain injuries associated with bleeding from large vessels and fracture of the skull.

2. The second type includes patients in whom the symptoms of compression occur after remissions. There are a number of types.

(a) One type particularly is associated with extradural haemorrhage from a ruptured middle meningeal vessel. At first the diagnosis is one of concussion, the patient proceeds to the stage of reaction and frequently becomes fully conscious. This period of full consciousness, in which the patient may walk about and be comparatively well, may last for a period varying from half an hour to twenty-four hours. The patient then becomes drowsy, various focal and localizing signs may appear, he becomes semi-conscious and then comatose. Such cases are often spoken of as "lucid interval cases." Though they are usually due to extradural haemorrhage, subdural haemorrhage may at times cause similar symptoms. On such patients definitely becoming unconscious, operation should be performed. It may be difficult to determine which side should be opened and sometimes it is necessary to operate on both sides. Paralyses, a single dilated and fixed pupil, haematoma, tenderness known to have been present in one temporal region, pitting on pressure in one temporal region or persistent copious bleeding from the ear are all valuable localizing signs.

(b) Some patients do not regain full consciousness, as just described, but at the stage of reaction only partly regain consciousness and then commence to lose consciousness again, becoming semi-conscious and comatose. They may show marked mental symptoms and may even be maniacal. The recognition of such conditions is difficult. The pulse rate slows and may pass to 40 per minute or less, whilst the blood pressure rises. It is during this stage that operation is indicated. Later, falling blood pressure and a rising pulse indicate that the patient has passed beyond the stage when intervention is of any value. Such cases are typical of an acute diffuse subdural haemorrhage from cerebral veins or large venous sinuses or severer forms of extradural haemorrhage.

(c) Some patients, after a stage of cerebral irritation, suddenly develop focal signs of a localized compression. The pathological lesion is a localized subdural haemorrhage associated with localized laceration and the acute symptoms are due to a recurrence of bleeding. Such patients may later present signs of cerebral tumour due to cyst formation or haematoma.

(d) Another group is uncommon and the condition often pursues a very long course extending over months. The patient may recover almost completely from the head injury, but headache, giddiness and slight mental symptoms are common. Later, symptoms of compression appear in a subacute form, paryses and other focal signs may appear and after a slow course the patient becomes comatose and dies. X rays may reveal a pneumatocele or at operation or *post mortem* examination a subdural cyst may be found.

(e) Late compression may also occur following traumatic intracerebral haemorrhage. The patient usually recovers almost completely. Rupture is apt to occur into a ventricle when acute apoplexy symptoms with high fever and fits develop, and the patient rapidly dies. When the rupture is on to the surface of the brain, the symptoms are very similar to those described in the previous type.

CONCLUSION.

Before concluding, it would perhaps be well to refer to the difficulties which may arise in the diagnosis between acute alcoholic poisoning, apoplexy *et cetera* and head injuries. In head injury the history and evidence of trauma usually make the diagnosis clear, but this is not always so, and it is wise to remember that patients who have taken alcohol, are more liable to encounter the causes of head injury than normal persons. Further, the dazing of patients following apoplexy or head injury may cause an apparent drunkenness and the smell of alcohol in the breath may be insufficient evidence of causation. In all cases of doubt patients should be kept under observation. In semi-conscious and unconscious patients, admitted without a history or definite signs of injury, besides alcoholic poisoning and apoplexy, the differential diagnosis must be made between head injury, poisoning by narcotic drugs and the comas of diabetes and uremia.

Finally, it may be said that in the present state of our knowledge it is difficult to diagnose accurately the exact pathological changes which take place after a head injury, as is nowadays our wont in many conditions. The most that we can do usually is to diagnose the general intracranial condition in much the same way that in the diagnosis of an acute abdominal condition a confident diagnosis of acute intestinal obstruction may be made with nothing further than a guess at its nature and site.

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SCIENTIFIC.

A MEETING OF THE SECTION OF NEUROLOGY AND PSYCHIATRY OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Mental Hospital, Callan Park, on July 17, 1930, DR. N. D. ROYLE, the Chairman, in the chair. The meeting took the form of a series of clinical demonstrations.

Mental Derangement and Disorder of Special Senses.

DR. T. M. ENGLAND showed four women suffering from mental derangement associated with deprivation of special senses.

The first patient was a woman, aged thirty-seven, married, with four children, all of whom were normal. There was nothing in her family history to note. She had married her first cousin and for months had been living

apart, attempting to support herself by doing domestic work. She had chiefly lived with her sisters and brothers, who found her irresponsible and difficult to control. She was a deaf mute, amenable, but somewhat irresponsible and childish and mildly euphoric. Her memory was moderately good. Her orientation as to time was uncertain. She was entirely unconcerned as to the past or future. In her physical condition there was nothing abnormal except that she was deaf and almost dumb. She could say a few words intelligibly, but usually wrote her replies to written questions. Dr. Clowes had reported that the nerve deafness had probably been acquired in early infancy. The nose and throat were normal. The *membrana tympani* on each side was normal.

The second patient shown by Dr. England was a married woman, aged sixty-eight with three normal children. There was nothing in her family history to note. She had been deaf and dumb since birth and was married to a deaf and dumb husband. She could read and write and could converse by her fingers. On admission to Callan Park she was very confused and depressed, and continually made the sign of cutting her throat. After she had been in hospital for some time she improved mentally, but still remained confused and disorientated as to time. Her physical condition was moderately good, except that she was mildly arteriosclerotic. Dr. Clowes reported that nose and throat were normal. The *membrana tympani* on each side was normal. Deafness was probably congenital.

The third patient was a single woman, aged thirty. In her family history it was noted that she had one epileptic sister who was deaf and who was a patient in Callan Park. She had one brother who was dull when young, but had improved. One sister was living at home; she was of a similar type to the patient, but less mentally weak. The mother was comparatively normal. The father was insane. This patient had not gone to school. She was not able to work continuously—she used to say she was too tired. She attempted to work in a boot factory for a time. She was obviously deficient mentally, but owing to deafness it was difficult to estimate the degree of deficiency. She was disorientated as to time. She knew her age. She was able to occupy herself usefully in the medical officers' quarters, although at times she became somewhat difficult to control. She could not read, but knew the difference between the appearance of words. She could hear loud noises, such as those caused by motor cars and bells. She was able to make sounds resembling speech, but what she said was mostly unintelligible. She was able to act as a useful messenger, and did not make mistakes in what she had to bring. Dr. Clowes had reported that deafness was probably congenital. Nose and throat and tympanic membranes were normal.

The fourth patient was a single woman, aged twenty-six, with nothing abnormal in her family history. She developed a paralysis in infancy and from the age of two and a half years had been "queer." She was at Waratah Convent for ten years, and lately had become what was described as "terrible" at home. She was generally a quiet patient, but occasionally was difficult to manage. She was correctly orientated as to time and said the hospital was "Rozella." She still retained persecutory ideas. She was in poor physical condition. She dragged both legs in walking, the left more than the right. The knee reflexes were active. Dr. Clowes had reported that deafness was probably acquired in early infancy. The nose and throat were normal and tympanic membranes were normal.

In the discussion which followed the demonstration of these patients, Professor W. S. Dawson queried the facilities that these patients might have had for speech education, and whether or not they might have benefited thereby.

Psychosis with Heart Disease.

DR. J. M. RAINBOW showed two patients suffering from psychosis with heart disease.

The first was a married man, aged fifty-three, a carter by occupation; he was a native of New South Wales. He had seven children, all normal. There was nothing

abnormal in his family history. He had been driving carts for thirty years. He gave no history of venereal disease or of heavy alcoholism. He was a moderate smoker. In May, 1929, he suffered from attacks of faintness with a suffocating feeling and a sense of restriction over the sternum. He went to the Royal Prince Alfred Hospital and was kept for three months in bed. His condition was diagnosed as disease of the coronary arteries. At Christmas, 1930, he quarrelled with his wife and had since lived apart. According to his own statements, "the children sided with her." He was living alone in a room until recently. He went to Liverpool State Hospital, but was unhappy there and decided to leave, but the medical superintendent said he needed to see a mental specialist, and he was sent to the Reception House. A statement from the medical superintendent at Liverpool was to the effect that the patient was afraid of being murdered while asleep.

The patient on admission was very depressed and had delusions that he was followed about by people who had designs on his life. He appeared to be of somewhat poor intelligence, but was able to give a correct account of himself. He was slightly deaf in both ears and had arteriosclerosis. The apex beat was diffused at the sixth intercostal space. The heart sounds were very poor in tone and had a fluttering, tic-tac rhythm. The blood had not reacted to the Wassermann test. After continuous rest in bed the patient became much brighter and was allowed to sit up. He soon, however, became very depressed and finally collapsed, lying completely exhausted, unable to reply to questions. After a further rest he again improved mentally, but still evidently retained his delusions of persecution. When he was allowed to get up and move about, he would again get the sense of impending harm, and finally collapse. This condition slowly improved, and at the time of the meeting he had lost his strong suicidal tendencies, but was still fearful, agitated and depressed.

The second patient shown by Dr. Rainbow was a man, aged twenty-five, single, and an invalid pensioner. There was nothing in his family history to note. His personal history was that at birth he was a premature baby, later a delicate child and a poor scholar. He worked till he was sixteen, but finally was unable to work on account of his heart, and lived with relatives. The only disease he had had was measles. His feet became swollen at times. The brother gave a vague history that the patient had been "strange for some time," that his heart had been weak since birth, and that he strained it at work. The patient suddenly and unprovokedly attacked a young woman in Redfern. He explained to the police that he was going to cut his throat, as he wanted to cure his heart trouble. On admission to Callan Park he was childish and irresponsible in manner. He made wild and foolish statements, and had no idea of where he was. He had not improved and remained sullen, morose, irritable and disinclined to talk. When asked how he felt, he shook his head and said: "No good." He was resistive to all attentions and often tried to kick and strike the attendants. He had tried to injure himself by throwing himself on the floor. He lay in bed most of the day, inert and uninterested. He was hallucinated and heard voices abusing him. He had a delusion that he was stabbed in the heart once with a sword. Physically he was a very thin, undersized, sallow youth, with a long, narrow face. He had a narrow, contracted thorax with anterior bulging. He was dyspneic. The apex beat was in the fifth intercostal space and the heart sounds were rapid, with a blowing mitral systolic murmur conducted into the axilla. The blood did not react to the Wassermann test.

In conclusion, Dr. Rainbow stated that it was questionable whether heart disease was more than an aggravating cause of the mental condition in the younger patient, who was probably suffering from *dementia praecox*. In the older patient it was a factor in producing the state of morbid apprehension, and his mental symptoms became more acute in proportion to the loss of compensation.

Psychosis and Paget's Disease.

PROFESSOR W. S. DAWSON and DR. J. M. RAINBOW showed a patient suffering from psychosis associated with Paget's

disease. The patient was a single man, aged thirty-seven, a labourer, working for the City Council, born in New South Wales. He had nothing of note in his family history. He was the youngest of five and the only son. He was at school till fourteen years of age, and then continued on at night school for three years. He became a cleaner for the City Council. He enlisted in 1916 at the age of twenty-two years and was two years in France. He suffered from "trench feet," but was not wounded. He seemed well on return and was discharged in 1920. He returned to work for the City Council. He had no history of venereal disease. He was apparently well until 1927, and then he began to get vague feelings that all was not well with himself. His "nerves" got bad, and he could not sleep owing to "all sorts of subjects running through his mind." He began to lose interest in affairs; he had previously been interested in politics. He believed he was being "doped" and had headache and several giddy turns, but did not actually fall. In September, 1927, his brain felt dull; before that it had been active all the while. His appetite failed. The action of his bowels was regular. He became religious, believing if men only had faith in the Almighty, they would be all right physically. He was not, however, keen on discussing this, and felt generally ill. He was a tall man of poor nutrition and physique, and was sallow. There was gingivitis and the pharynx was hyperemic. He was slow and lethargic in movements. His eye lids tended to droop. The right fronto-parietal area of the skull bulged prominently. The temporal arteries were tortuous and pulsated visibly. His heart was not enlarged. A soft mitral systolic murmur was present. The arteries were palpable. The systolic blood pressure was 130 and the diastolic pressure 60 millimetres of mercury. The breath sounds were harsh and vesicular, with some prolongation at the apices. No abnormality was detected in the abdomen. The urine contained a heavy deposit of phosphates, but no other abnormal constituents. The response of the right pupil was brisk and of the left sluggish. The superficial reflexes were present. The knee jerks were sluggish. No Rombergism was present and no incoordination and no tremor were noted. The Wassermann test yielded no reaction. In regard to his mental condition, the patient talked "fairly rationally," but was apt to launch off quite unexpectedly, for example, when he admitted his religious ideas. He made grimaces and clasped and unclasped his hands. A friend stated that the skull deformity was of "long standing," but seemed to show up more now that the patient had got thin. He had always been eccentric. Lately he was fond of a young woman above his social and mental level. He told the friend he had been treated at Sydney Hospital lately for syphilis and this was worrying him. He was admitted to Callan Park on November 24, 1927, as a voluntary patient. At this time he appeared stuporous and confused. He was faulty in habits and needed help in dressing and feeding.

Since admission he had remained confused. At the time of the meeting he was mute and took little notice of anything. The apparent bulging of the frontal bone had slightly increased. The tibiae manifested anterior curving. The spinal column was curved.

In the discussion which followed the demonstration of this patient, Dr. A. T. Nisbet explained that the thickening of the skull, which was well shown in the skullgram, was not syphilitic, that in syphilitic conditions the bone thickening was laminated, whilst in Paget's disease there was a "sun ray" appearance. He also stated that Paget's disease was more common than generally supposed; he was not surprised to see it in this man of thirty-seven and had recently seen it in a man of twenty-five.

Syphilitic Encephalitis.

Professor Dawson also showed a patient who was suffering from syphilitic encephalitis. The patient was a man, aged thirty-five, who had been under his care in the Royal Prince Alfred Hospital. The patient was admitted in a state of acute confusion; he was unable to give a reliable account of himself, but a friend reported that he had complained of his eyes and had displayed loss of memory for some weeks. He was in a state of mild cerebral

irritability, restless and complaining of pain in his head and of stiffness at the back of the neck. Examination was not tolerated well. There was paralysis of the external rectus of the left eye, with slight ptosis. The pupillary, cutaneous and tendon reflexes were all brisk. Sensation appeared to be intact. The blood and cerebro-spinal fluid gave a positive reaction to the Wassermann test. There were ten cells per cubic millimetre and a slight increase in globulin in the cerebro-spinal fluid, which was under increased pressure. At times he was in a state of fear and imagined that his life was threatened and that men were waiting for him outside the window. On one occasion he afforded some amusement to other patients by submitting to drill and by carrying out absurd actions to command. One day the headache was specially severe and he vomited twice. He remained in a state of confusion, aurally hallucinated, disorientated as to time and place, mistaking identities and displaying the suggestibility of Korsakow's syndrome for over a month. He received three weekly injections of "Muthanol," 0.2 grammes, and "Novarsenobillon," 0.6 grammes, but his restlessness and confusion were such that he could no longer be nursed in a general hospital. He was admitted to Callan Park Mental Hospital towards the end of June and then became quieter and clearer in mind, so that he could give a connected account of his illness.

Professor Dawson pointed out that the ocular palsies persisted. Antisyphilitic treatment was being continued. A recent examination of the cerebro-spinal fluid showed that the Wassermann reaction was still positive and the bicolour reaction was of the paretic type.

Tremor of the Arms.

DR. C. HENRY showed a man, aged fifty-one, a journalist by occupation, with a condition that had been provisionally diagnosed as functional tremor of the arms. It was noted that in his family history his father had a slight tremor of the right arm, but could control it so that it was not noticeable. His parents were natives of France. He was a healthy youth. He had a university education. He enlisted in 1915 and suffered from severe shell-shock. Since then he had had a tremor of the right arm which interfered with his earning capacity, and he had periods of acute mental depression associated with alcoholism. There was nothing in his history to suggest that he had had encephalitis. His arteries were healthy. His heart was normal, and his systolic and diastolic blood pressure 125 and 80 millimetres of mercury respectively. There was no nystagmus or scanning in speech. When he attempted to grasp an object, such as a pen, there was a slight, coarse tremor, which increased definitely after grasping, so that writing was almost impossible, and a cup could not be held without its contents being spilled. Attention to this increased the tremor, but when his attention was distracted, such as when playing golf, there was no tremor. Under alcoholic stimulation the tremor temporarily disappeared, but was much increased afterwards. There was no pronounced spasticity, except cogwheel resistance to passive movement. His arms swung freely when walking and there was no evidence of Parkinsonism.

In the discussion which followed, Dr. S. Evan Jones asked for the diagnosis and pointed out that the tremor did not resemble that of typical *paralysis agitans*. He considered that the condition was functional.

DR. G. L. O'NEILL considered that, as the deep and superficial reflexes were all exaggerated, there was an organic factor.

DR. J. A. L. WALLACE mentioned that dipsomania was an accompanying factor in this case and considered it probable that the two conditions were related.

Post-Encephalitic Parkinsonism.

The second patient shown by Dr. Henry was a single man, aged thirty-one, with post-encephalitic Parkinsonism. He had been a healthy child and youth. He enlisted in 1914 and had a severe illness on active service at Rouen in 1918, which appeared to have been encephalitis. In 1919 he was admitted to Belle Vue Hospital, New York, with "meningitis." In 1920 and each succeeding year he was in a hospital with slowly increasing signs of

Parkinsonism. In this case the following signs of Parkinsonism were well marked: Difficult phonation, monotonous speech, micrographia, slow shuffling gait, inability to stop quickly, *kinesia paradoxica*, reduced arm swinging, tendency to spurious hilarity with lingering Parkinsonian smile, definite tremor of the right hand, temporary voluntary inhibition of tremor, infrequent blinking, ocular fixity, impairment of ocular convergence, cogwheel rigidity. The patient's condition was improving with stramonium.

A Case for Diagnosis.

The third patient shown by Dr. Henry was a patient with a neurological condition for diagnosis. The patient was a married woman, aged forty-seven, with four children. There was nothing abnormal noted in her family history. In her personal history she was stated to have had "wasting disease" in infancy. She was a good musician and scholar and married at thirty-one. Four years ago her husband lost his job and she had to go to work. She overworked and could not sleep and had "never been well since." She was always very introspective and worried over her condition, on which she was ready to talk at length. She said: "I have to hold my mouth to one side, due to want of rest." The abnormalities she presented were: Convulsive movement of trunk and head, which ceased during sleep; left-sided facial paresis; paresis of interosseous and lumbrical muscles of both hands; greatly exaggerated knee jerk; right-sided patellar clonus; extreme exaggeration of the plantar reflex on both sides.

In the discussion, Dr. S. Evan Jones recalled that when this patient was in Broughton Hall four years before, she spoke from one side of her mouth only and stated that she did this to prevent something getting in the other side. He regarded her condition as purely functional.

DR. N. D. ROYLE suggested phrenicotomy to relieve the spasmodic contraction of the diaphragm. He thought the condition had an organic basis.

PROFESSOR W. S. DAWSON suggested that there was a degeneration, involving the basal ganglia, but admitted that the condition presented many unusual features.

DR. HENRY in reply said that he was frankly puzzled about the case, but had always held the unusual opinion that all so-called functional conditions had an organic basis, a view that was gaining ground elsewhere, especially in view of Pavlov's remarkable studies of the conditioned reflex. In this patient all the deep (tendon) reflexes were exaggerated. Was this consistent with a "functional" disorder?

Post-Encephalitic Condition.

DR. HENRY'S fourth patient was a single girl, aged twenty, with a post-encephalitic condition. There was nothing in her family history. She had a normal childhood until twelve, when she had encephalitis with diplopia and lethargy. No immediate ill effects occurred and she was able to play tennis and golf. At the age of sixteen she showed signs of rigidity and tremor which had slowly progressed since. Mentally she showed a certain amount of post-encephalitic delinquency—she was violent to her mother, quarrelsome, querulous, and teased other patients. Her whole mental and physical condition was that of a typical Parkinsonian syndrome.

Senile Dementia.

DR. J. A. L. WALLACE showed an elderly man, aged seventy-one, an invalid pensioner, with *angina pectoris*. This patient was a married man with a family, who was sent to Callan Park through the Reception House, on account of having threatened his family, with the delusion that they desired to rob him and persecute him; he had also the morbid idea that his fainting fits were caused by the persecutions of his family. The only point in his family history noted was that his father died at forty-eight of "consumption." In his personal history it was stated that he had been a weakly child, but a good scholar. He followed school by training in a marine officers' training college. After four years in a sailing ship he passed through various grades as marine officer on steam liners. He stated that he had suffered from malaria, syphilis,

cholera, Chagres fever, jungle fever and *angina pectoris*. He was moderate in his habits as regards alcohol and tobacco, but immoderate in his sexual indulgences, and, in his own words, was "often with harlots in South America." The patient was in a mental hospital for a short time in Buenos Ayres. He said he was a "little off his head" with financial worry. Physically he was pale and sallow. His systems were normal, except in his circulatory system. He had arteriosclerosis with a systolic blood pressure of 175 and a diastolic pressure of 95 millimetres of mercury. A skilogram of the thorax showed a dilatation of the right side of the descending aorta. His blood did not react to the Wassermann test. His mental condition was one of mild senile deterioration, with the usual persecutory type of idea. He was unable to realize that his difficulties with his relatives were due to his own conduct. He was facile and became childishly loquacious. While in hospital the patient had given no difficulty in management. Dr. Wallace said that his wife's statement would give some idea of the difficulties of having such a patient at his own home. She wrote that, some years ago, he developed "strong signs of insanity" without apparent cause, with religious mania. His local doctor told her then that her husband's "brain was falling to pieces." He then got ideas in regard to poisoned food, secret societies *et cetera*. This condition lasted three or four years. Then five months ago he became "queer" again, and evidently residents in the neighbourhood complained of his going to their houses and quarrelling and wanting to fight. He was considered to be "eccentric" for years.

Skiagrams.

DR. A. T. NISBET, from the X ray Department, Mental Hospital, Parramatta, showed a number of radiographs illustrating physical disorders present in patients who were mentally ill.

Of an interesting group of melancholics who had developed marked confusion, three had early tuberculous infiltration of the lung, one had primary gastric carcinoma and secondary deposits on the liver, two had infection of the maxillary antra and teeth, the remaining two being sufferers from megacolon. One of the latter had had a sympathetic ramisection performed by Dr. Royle and had improved remarkably.

Of another group of epileptic patients, each had marked visceroptosis.

Dr. Nisbet also showed an interesting group of radiographs of the skull, including the case of Paget's disease already described, two cases of Fröhlich's syndrome, a case of acromegaly and various other interesting skulls.

Medical Societies.

THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN, BRISBANE.

A MEETING OF THE CLINICAL SOCIETY OF THE HOSPITAL FOR SICK CHILDREN was held at the Hospital for Sick Children, Brisbane, on July 24, 1931, DR. D. GIFFORD CROLL, the President, in the chair. The meeting took the form of a series of demonstrations by members of the honorary staff.

Cretinism.

DR. K. B. FRASER showed a female child, aged four years, whose mental development corresponded to that of a child of one year. She was short and thick-set. Her skin was parchment-like; it was loose and redundant and puckered easily on the forehead. There was puffiness between the eyes and the expression was dull. The child was always lethargic. She was not irritable and was easily amused. The head was dolichocephalic. The hands were broad and short. There was an umbilical hernia. The child was undoubtedly suffering from cretinism. Unusual features, however, were the abundant growth of fine wavy hair and the condition of the skin, which was smooth and soft.

The child had been treated by means of thyroid extract on several occasions, but the dose had always been too small and the treatment had never been continued for a sufficiently long period. Dr. Fraser stressed the necessity of recognizing the condition when the child was yet very young, so that the maximum of benefit might be possible from appropriate treatment.

Congenital Syphilis.

DR. S. F. McDONALD showed a well grown male child who, during the previous eighteen months, had become clumsy with his hands and had had some difficulty in walking. Dr. McDonald had first seen him four months before the meeting. The hands were then very wasted; there was atrophy of all the essential muscles of the fingers. The calves were wasted and the feet were in the position of *pes cavus*. An indefinite Babinski sign was present in each foot. The knee jerks were exaggerated. The right ankle jerk was indefinite, the left was present. The abdominal reflexes were sluggish. The tongue had the peculiar appearance known as "worms in a bag." There were no eye changes and no alterations in sensation. There was no actual ataxia.

There was nothing of importance discovered in the family history. The blood reacted to the Wassermann test.

Treatment by means of inunctions and injections of "Neokarsivan" had been followed by some improvement.

Dr. McDonald remarked that the outlook was much better for this child than for the ordinary congenital syphilitic. It was questionable whether he would recover the use of the atrophied muscles, but the disease would be arrested. Clinically the symptoms were suggestive of progressive muscular atrophy.

Celiac Disease and Congenital Syphilis.

Dr. McDonald's second patient was a female child, aged four and a half years, who had been attending as an outpatient for a period of twelve months. She was intolerant of fat. Her blood reacted to the Wassermann test. Before her admission to hospital she had the typical appearances associated with celiac disease—the pot belly and shrunken buttocks. She had received antisyphilitic treatment, but improvement had not been very satisfactory. She was admitted to hospital, as it had appeared likely that the instructions regarding her diet were not being followed at home. During her stay in hospital she was given an ordinary fat-free diet; at the time of the meeting she looked well.

Dr. McDonald pointed out that the most important part of the treatment for these children was the elimination of fat, particularly milk fat, from the diet. Skimmed milk should be used. These children frequently did not like fruit and vegetables, hence they had been known to become affected with scurvy.

Chronic Nephritis.

Dr. McDonald next showed a male patient, aged ten years, who, five weeks before the meeting, had fallen from a balcony, had vomited a good deal throughout one day and had then been stricken with a convulsive seizure. Further symptoms were headache and delirium. It was at first thought that he was suffering from either concussion or cerebral haemorrhage, but there was a large quantity of albumin in the urine and the pulse was found to have a high tension. The systolic blood pressure was 210 and the diastolic 180 millimetres of mercury. The specific gravity of the urine was 1.004; the urine contained pus and red blood cells, hyaline casts and a few granular casts. The blood urea content was 129 milligrammes per 100 cubic centimetres. There were 4,170,000 red blood cells and 16,000 white cells per cubic millimetre of blood; the percentage of haemoglobin was 82 and the colour index was 1.0. There was no punctate basophilia. Ophthalmoscopic examination revealed haemorrhages and exudate round the discs. The child did not pass any urine at night.

Dr. McDonald remarked that the fall had evidently been due either to a mild uræmic attack or to a small cerebral haemorrhage. There was no history of lead poisoning and

the mother denied that the child could have had access to lead.

At the time of the meeting the blood pressure had fallen to 160 millimetres of mercury, systolic, and 130 diastolic over a period of ten days. Treatment had been by means of hot packs and rectal injections of a strong solution of magnesium sulphate.

Tuberculous Adenitis.

On behalf of Dr. G. P. DIXON, Dr. S. F. McDonald also showed a female child, aged ten years, who had been admitted to hospital on July 7, 1930. She had attended the out-patients' department for a period of twelve months suffering from a lump on the right side of the neck, in the anterior triangle. It had never been painful; it had become larger and softer shortly before admission. At the time of admission the swelling was observed to be red and fluctuant. Aspiration revealed the presence of pus. Tubercle bacilli were found. The application of the von Pirquet test resulted in a violent reaction.

The use of the mercury vapour lamp was suggested for treatment. Removal of tonsils had been suggested, but Dr. McDonald thought that this procedure might not be wise in view of the existence of the tuberculous infection.

Sarcoma of the Orbit.

Dr. E. O. MARKS showed a child, aged four years, who had been under treatment in hospital one year previously. On October 19, 1928, Dr. Lockhart Gibson had removed a myxosarcoma, the size of a small walnut, from the upper and inner quadrant of the orbit. Five milligrammes of radium had then been applied and left in position for seventy hours. The eye had remained unaffected. On January 3, 1929, the patient had been seen again by Dr. Gibson, who had informed the parents that there appeared to be a recurrence. At the end of January, 1929, Dr. Graham Brown had reported that the eye was proptosed. Radium had been applied and the condition had appeared to improve somewhat. In June, 1929, the child had been admitted to hospital and had been seen by Dr. Gibson in consultation. At this time it was observed that the whole eye was proptosed and very hard. At operation, which was performed at once, the whole contents of the orbit were removed. Dr. McDowell placed one milligramme of radium in the optic canal, five milligrammes into the orbit and five milligrammes round the orbit.

At the time of the meeting there had been no recurrence, but the orbital cavity was filling up. Dr. Marks said that this would have to be carefully watched in view of the possibility of recurrence.

Cardiac Failure.

Dr. GAVIN CAMERON showed a male child, aged eleven years, who had been admitted on July 21, 1930. He had tired readily and had suffered from attacks of nausea and, occasionally, vomiting. He had been worse during the week prior to his admission and had suffered from breathlessness. He had been operated on in January, 1930, for the relief of abscess in the muscles of the right side of the chest; a systolic murmur had been noted at this time and the liver border had been palpated at a distance of 2.5 centimetres (one inch) below the costal border.

At the time of his admission he was in good general condition. The apex beat was in the sixth intercostal space, 11.75 centimetres (four and a half inches) from the mid-sternal line. There was a systolic murmur at the apex and at the pulmonary orifice. The pulmonary second sound was reduplicated. The liver was tremendously enlarged, the spleen was not palpable. There were enlarged glands in the groin and axilla and neck. No abnormality save cardiac enlargement was revealed by radiological examination. There was no pyrexia. The red blood cells numbered 5,000,000 and the white cells 14,000 per cubic millimetre. The haemoglobin percentage was 110 and the colour index was 1.07. The types of leucocytes were in normal proportions. The urine contained a trace of albumin, but no abnormality was detected by microscopical examination. There was some ascites. There was no reaction to the Wassermann test or the complement fixation test for hydatid.

Dr. Cameron asked whether the enlargement of the liver was due to congestion, the result of the failing heart.

Osteomyelitis.

Dr. C. TUCKER showed a girl, aged six years, who had been admitted on February 7, 1930. Two weeks before admission she had become affected with pain in the lower part of the right leg; multiple incisions had been made into the leg by a medical practitioner in the country. One week prior to her admission to hospital an incision had been made into a swollen area over the left scapula. At the time of her admission to hospital she was tender to pressure over the right tibia, the left femur and the left scapula. There were many discharging wounds.

An incision was made over the lower end of the right tibia. At a point 5.0 centimetres (two inches) from the lower end of the tibia there was a perforation in the bone, through which pus was exuding. The diaphysis was almost completely separated from the lower epiphysis and the cancellous bone was very soft. The incision in the scapula was enlarged; the neck of the scapula was found to be affected with osteomyelitis.

There was considerable pyrexia, the child was very ill and became very anaemic. There was no involvement of the kidneys.

In March, 1930, the distal epiphysis of the right tibia became affected. At operation it was gouged out and was found to be a mass of mushy tissue. During the same operation it was observed that the scapula was honey-combed with small holes filled with mushy tissue. Irrigation with Dakin's solution, "Eusol" and hypotonic saline solution was employed. On April 1, 1930, the leucocytes numbered 6,900 per cubic millimetre and the red blood cells 2,500,000 per cubic millimetre; the haemoglobin percentage was 26 and the colour index 0.6. There was irregularity in size and shape of the red cells. There was polychromasia, but no nucleated red cells were present. A transfusion of blood was given. Pyrexia was of the swinging type until the end of May. The child then spent four weeks in a convalescent home and a further two weeks in hospital. At the time of the meeting her condition had improved greatly.

Dr. Tucker remarked that the formation of sequestra was uncommon after guttering operations such as those he had performed on this child.

Septic Arthritis.

Dr. Tucker's second patient was a girl, aged one year and eight months, who had been admitted to hospital on June 15, 1930. Her knee had been caught in a picket fence and two days later had become swollen. At the time of admission the joint was swollen and red. On June 16, 1930, pus was aspirated from the joint. An incision was then made on either side and much pus was evacuated. Daily irrigations were carried out. Progress during the following four days was satisfactory, but a week thereafter the temperature was raised above normal and it was found that the incisions had closed. The wounds were reopened and irrigation recommended. There was a tendency to backward dislocation of the tibia. At the time of the meeting the leg was flexed at the knee and could not be extended.

Dr. Tucker asked for an opinion as to what should be done.

Post-Graduate Work.

ANNUAL POST-GRADUATE COURSE IN BRISBANE.

THE Annual Post-Graduate Course will be held in Brisbane from June 1 to 5, 1931.

Dr. B. T. Edye, Dr. Herbert Schlink and Professor C. G. Lambie, of Sydney, will deliver lectures and give demonstrations on various subjects.

Professor F. Wood Jones, of Melbourne, will, on Friday, June 5, deliver the Joseph Bancroft Memorial Lecture, on a subject to be notified later.

Members are asked to notify the Joint Honorary Secretaries, Dr. Keith Ross and Dr. Neville G. Sutton, British Medical Association Building, 35, Adelaide Street, Brisbane, that they intend to be present and whether or not they will be accompanied by their wives.

ANNUAL REFRESHER COURSE IN MELBOURNE.

THE Annual Refresher Post-Graduate Course arranged by the Melbourne Permanent Committee for Post-Graduate Work will be held this year from August 17 till August 29 inclusive. Full details of the course will be announced subsequently. The fee for this course will be £3 3s.

Arrangements have been made with the Melbourne and Alfred Hospitals for the accommodation of a limited number of graduates at a charge of three guineas per week. Early application to the Honorary Secretary of the Post-Graduate Committee for this accommodation is requested.

LECTURES IN BACTERIOLOGY AND IMMUNOLOGY.

THE Melbourne Permanent Committee for Post-Graduate Work has arranged for Dr. F. M. Burnet, Assistant Director, Walter and Eliza Hall Institute of Research, Melbourne, to deliver a course of six lectures on bacteriology and immunology during the currency of the refresher course. The fee for the course will be £2 2s.

The syllabus is as follows:

Lecture I: The Basis of Immunology.—The antigen-antibody reaction; toxins and antitoxins; demonstration of staphylococcus toxin as a typical antigen; methods of detecting and measuring antibodies and their biological significance.

Lecture II: Immunological Aspects of Bacterial Infections.—Modern work on bacterial structure; the specific carbohydrate antigens of bacteria; the nature of virulence and the mechanism of specific serum therapy in bacterial infections, particularly with reference to pneumococcal infections. The structure of the intestinal bacteria; modern developments of the Widal reaction. Bacterial vaccines. Endotoxins.

Lecture III: Immunological Aspects of Bacterial Infections (continued).—Bacterial toxins; the Schick and Dick tests; the methods of producing antitoxic immunity; anatoxins. Bacteriophage, its possible significance in intestinal and urinary infections.

Lecture IV: Modern Work on Epidemiology.—Experimental epidemiology; the mouse epidemics of Topley and Webster and their bearing on human epidemiology; the significance of carriers and of subclinical immunization.

Lecture V: The Filterable Viruses.—General survey of the types of human disease caused by filterable viruses. The neurotropic viruses and the problems of encephalitis. The Rous sarcoma. Immunity to filterable viruses; the therapeutic use of convalescent serum.

Lecture VI: The Modern Theory of Anaphylaxis.—The cellular theory; the significance of histamine according to Dale and Lewis. Non-specific protein shock and its therapeutic uses. Bacterial allergies.

It is requested that those proposing to attend these courses will send their names and fee as soon as possible to the Honorary Secretary, Melbourne Permanent Committee for Post-Graduate Work, 12, Collins Street, Melbourne, C.I.

Obituary.

MORRIS FREDERICK HORSLEY GAMBLE.

DR. MORRIS FREDERICK HORSLEY GAMBLE, whose death, as recorded in a recent issue, occurred on February 23, 1931, was born in Prahran, Victoria, in 1865. He was the son of the late Walter Mitchell Gamble, sometime Chief Inspector of Schools in Victoria. He went to school at the

Punt Road State School and at the Melbourne Grammar School. He began his study of medicine at the University of Melbourne and completed his undergraduate course in 1896 at the University of Edinburgh, where he became licentiate of the Royal College of Physicians and the Royal College of Surgeons. After practising for three years in Edinburgh he returned to Australia and in 1900 was appointed Resident Medical Officer of the Hospital for the Insane at Sunbury. His promotion in the Lunacy Department was rapid and in 1910 he became Superintendent of the Mental Hospital at Kew. He remained here until 1923, when he left the Lunacy Department and started practice as a consultant in Collins Street. In 1925 he was appointed Honorary Consultant Psychiatrist to the Melbourne Hospital and also became Honorary Psychiatrist to the Alfred Hospital; here he did valuable work in the establishment of the Psychiatric Clinic. Gamble kept up his reading and was called upon on several occasions to give post-graduate lectures in neurology and mental diseases. He was for some years associated with Mr. A. A. Kelly in the study of juvenile delinquency.

Gamble's main hobby was drawing and for many years, particularly in the early stages of his career, he was a contributor of black and white caricatures and sketches to *The Bulletin*, *Punch* and other papers. Even in his student days at Edinburgh he was a regular contributor of cartoons to several weekly papers. He was highly esteemed by his colleagues. They recognized the breadth of his outlook and the soundness of his judgement. His place will not be easily filled. He leaves a widow and six children, one of whom is the widow of the late W. A. T. Lind, well known for many years as a contributor to this journal.

Dr. W. Ernest Jones writes:

The sudden and regrettable death of Dr. Gamble calls for something more than a mere reference to the passing of a respected medical practitioner, something in the nature of a well earned eulogy.

Morris Frederick Horsley Gamble was educated at the Melbourne Grammar School. He proceeded first of all to the University of Melbourne and then to Edinburgh, taking the qualifications of the colleges in 1897. After practising in the Old Country, he returned to Victoria, and in 1899 he joined the medical staff of the Lunacy Department, his first appointment being at the Sunbury Hospital for the Insane. He was subsequently transferred to Kew where he was the medical officer in charge on the female side.

It was at this time that the writer made his acquaintance and was able, in the reconstruction of the department, to appoint Gamble as Senior Medical Officer at that institution. His work at that time was characterized by thoroughness and enthusiasm, and he well deserved his subsequent appointment as Medical Superintendent at the Hospital for the Insane at Ararat, to which position he was gazetted on May 1, 1908. Two years later he was transferred to a similar position at Kew and he there commenced his work of administering that institution, which for a period of nearly twelve years he prosecuted with the same care and judgement as he had exhibited previously.

He lectured to the medical students and also gave post-graduate instruction in a characteristically conscientious manner; his sound knowledge of psychiatry exhibited itself by the admirable manner of his reports and his presentation of the cases of the patients under his care.

He took pride in the fact that at Kew he had entirely obliterated the use of restraint and reduced seclusion to a minimum, no mean task, considering the difficult type of patients he controlled.

He was an enthusiastic supporter of the Clouston school and a scathing critic of the psycho-analytic teaching emanating from Vienna.

On his resignation in 1922 he left the Department to take up consultant work in Collins Street and was followed by the good wishes of all the members of the staff at Kew and the respect of all his colleagues.

Since that time he identified himself with every forward movement to improve this branch of the medical art. He was appointed as Psychiatrist to the Alfred Hospital

and Honorary Consultant in Mental Disorders to the Melbourne Hospital. He supervised the work of certification of the patients in those hospitals with his usual care and conscientiousness. He devoted a considerable amount of his time to the subject of juvenile delinquency and supplied to the magistrates of the children's courts many carefully considered opinions as to what should be done with the problem children who were constantly coming before the courts.

Morris Gamble was a man who never sought the lime-light. He was essentially a worker and a family man, never happier than when at home. He contributed a few and always very sound articles to our Australian journals on subjects within his specialty, and he had a charming gift of caricature which unfortunately he made too little use of.

His sudden death will be regretted by a very wide circle of friends, patients and colleagues.

CHARLES RICHARD PLAYER.

We regret to announce the death of Dr. Charles Richard Player, which occurred on March 23, 1931, at Malvern, Victoria.

Correspondence.

SPIKED BY A PLATYPUS.

SIR: Dr. J. Billing Halford sent me the following interesting description by the late J. W. Rusden (1874):

A native opened a platypus and in doing so received a wound on the wrist from the hind feet in its death struggles. He felt faint and had to rest. Within an hour he showed the swelling at the puncture areas. The arm, especially at the shoulder, was much swollen for many days and the swelling of the gland so painful that the arm was useless for some time. For some weeks it seemed to be undergoing atrophy. Eventually it regained its tone. Dr. Thompson, Walter and Eliza Hall Institute, informs me that the animal only strikes when its ventral surface is irritated.

Yours, etc.,

W. KENT HUGHES.

22, Collins Street,
Melbourne,
March 2, 1931.

THE VERNES TEST AS APPLIED TO SYPHILIS.

SIR: Dr. Woodruff's paper of February 28 makes exceedingly interesting reading. But it is difficult to reconcile his statements with the table published at the last "League of Nations Report of the Conference on the Serodiagnosis of Syphilis": "Professor Vernes and M. Bricq, Syphilitic Method." "Table showing the results obtained by Professor Vernes, compared with the corresponding results obtained by other methods." Four hundred and forty-eight sera from syphilitic cases gave, by ten other methods, an average of 231 positive findings (+++), whereas Vernes shows 171 positive findings of the same degree.

Yours, etc.,

A. E. FINCKH.

227, Macquarie Street,
Sydney,
March 5, 1931.

OSTEOMYELITIS.

SIR: In your useful, informative and critical articles described as "Current Comment" you have shown in this week's issue of the journal how diverse and unsettled are

the opinions of surgeons in the treatment of osteomyelitis. This want of standardization in the treatment of a special variety of abscess in spite of the lapse of more than fifty years since the means of cure were made available to us, is not only amazing, but decidedly disconcerting and opprobrious. Of the many methods you mention, prominence is given to that recommended by H. Winnett Orr. This surgeon is honest enough to admit that his technique is founded on no principle, that it is in fact entirely empirical. Can it be worthy of consideration under these circumstances? Empiricism is justifiable to the nescient. Do we admit inclusion in that category? It may be helpful to remind you and your readers that the only method of treating abscesses connected with bone, which you have not referred to, is that recommended by Lister.

Tradition and contemporary record, not only Lister's, prove that such cases were treated with astonishing success. In fact, it is claimed that: "There was no greater glory in the history of antisepctic surgery" than in the results of treatment of this class of case.

Would it not be worth while, in view of the revelations so well epitomized in your article, to adopt Lister's principle in the next series of cases and so attempt to establish some kind of standardization in the management of diseases associated with abscess of bone?

Yours, etc.,

A. C. F. HALFORD,
M.D. (Melb.), F.C.S.A.

Wickham House,
Brisbane,
March 7, 1931.

TRACHOMA.

SIR: Some little time ago I wrote to you on the subject of trachoma with special reference to its contagious nature. I have just received from Egypt the annual report of the Giza Memorial Ophthalmic Laboratory in which the following statements appear.

It was found impossible to inoculate monkeys with trachoma. A folliculosis common to monkeys appeared, but no true trachoma. That inoculations with a small Gram-negative bacillus isolated from trachoma equally failed to produce trachoma, though the writer admits that trachoma in monkeys may not be the same in character as that in man.

It is pointed out that Piringer successfully inoculated one patient with the discharge from another and by his inoculation experiments on the human species has shown that the various clinical forms of the disease are identical.

Doubt has, however, been expressed as to whether it is possible to convey trachoma in the absence of some other form of conjunctival infection. In other words, a symbiosis may be necessary.

This experimental investigation cannot be regarded as final, but it is significant that in Australia most cases of trachoma are found in hot, dry and dusty districts where conjunctivitis is common.

There is something we do not fully understand about the causation of gonorrhœal ophthalmia, of trachoma, and of leprosy. It is quite evident that in all three cases there is a factor in addition to simple inoculation still to be disclosed.

Yours, etc.,

JAMES W. BARRETT.

103-105, Collins Street,
Melbourne,
March 11, 1931.

TEETH EXTRACTION.

SIR: May I support Dr. R. N. Burton's letter of February 2 concerning the ordering of wholesale extraction of teeth on insufficient grounds?

I think the root of the evil may be found in the medical curriculum. Today the student is given instruction in ophthalmology, diseases of the ear, nose and throat, radiology and other special branches of medicine and surgery, but the study of oral diseases is entirely neglected. At most he is taught to examine the mouth in cases of infectious fevers, but even then, if he has never been trained to observe the normal, how can he appreciate the abnormal? I have known a student, in writing a case history, to state that "bad pyorrhœa" was present, and yet on subsequent examination it was found that the patient was wearing (certainly very dirty) full upper and lower artificial dentures. Many medical graduates can make a skilful examination of the tonsils, naso-pharynx and larynx, and after having done so, can arrive at a very sound conclusion concerning the condition of those parts, but are they not often, as Dr. Robert Hutchison observed, "trying to see beyond the brick wall without looking on this side of it"?

As Dr. Burton suggests, a slight gingivitis is often diagnosed as pyorrhœa by the medical practitioner in the same spirit of ignorance that the presence of the lingual tonsil is quoted as evidence of incomplete removal of its faecal neighbour. Without the competent opinion of a dental surgeon, arrived at after careful clinical and X ray examination, it is unwise to sacrifice teeth on the assumption that they may be the source of a septic focus. The decision to condemn a patient to wear artificial dentures for the rest of his life is not a thing to be lightly considered, especially as it is often impossible to ascertain beforehand whether the alveolar process which remains after extraction of the teeth will be suitable for the support of mechanical substitutes.

Yours, etc.,

ARTHUR AMIES,
L.R.C.P., L.R.C.S. (Edinburgh), L.D.S. (Victoria),
D.D.Sc. (Melbourne).

32, Collins Street,
Melbourne,
March 11, 1931.

A WORKERS' COMPENSATION CASE.

SIR: A case recently before the Workers' Compensation Commission presented some points of interest so far as the medical evidence given at the hearing was concerned.

A labourer commenced work for an employer on a Monday and discontinued on account of sickness on the Thursday following. He was admitted to hospital on the following day and died five days later, the cause being given as pleurisy and double pneumonia.

It was claimed that he received a wetting each day he worked, but this was questioned as regards the first three days. It was admitted, however, that he worked in water on the Thursday for about four hours.

He was said to have been flushed in the face on Monday evening, to have complained of pain in the side of his chest on Tuesday, and to have had a heavy cold in his head from that day to the time he knocked off on Thursday. This meagre history can be taken to indicate that he had an acute infection of his upper respiratory tract followed later by pneumonia. Opinions were expressed that the pneumonia commenced when the costal pains were complained of on Tuesday, but it is difficult to believe that he could have worked for two days after the onset of pneumonia. Osler, quoting Traube, says that often definite prodromes may occur and last for several days or even weeks; also that the patient often has a laryngitis, coryza or bronchitis for some time before the onset of pneumonia.

The medical witnesses for the applicant were not prepared to state that the conditions of exposure to wet the man was subjected to at work caused the pneumonia, but they satisfied the Commission that the wetting on Thursday aggravated the illness to a degree sufficient to indicate it as the determining factor in the fatal termination of the case, that is, if the man had not been so exposed, he would have recovered.

Now it seems to me that this is an absolutely unwarranted assumption, even though the opinion was skilfully drawn out of them by the specious questioning of counsel, who asked if they would recommend a patient suffering from pneumonia to expose himself to such wetting and whether they would expect such exposure to make a man in this condition worse or better.

If the man had recovered, would these same witnesses or any other medical men feel in a position to state that the illness was aggravated in intensity or prolonged for one week, one day or even one minute by reason of the work the man was doing in the wet during the incubation period, the prodromal period or even the early stage of the disease? And if they cannot say it would or cannot give any valid reasons for an opinion that it would, how can they hold that in this particular case it was the factor that determined the fatal ending? Are they not placed on the horns of a dilemma?

Yours, etc.,

E. S. STOKES.

341, Pitt Street,
Sydney,
March 17, 1931.

POLIOMYELITIS.

SIR: In the Journal of March 14, 1931, the following statement is credited to Dr. Jean Macnamara: "The usual story, the common story, in 1898 and 1908 was as follows. A child, previously well, would go to bed well and be found in the morning with a paralysed limb, but in the last five years in Australia and in America this had not occurred."

My records have been kept since 1920 and out of three hundred and eighty patients' histories a history similar to that mentioned by Dr. Macnamara has been given in forty-six instances, or 12.1%. In a number of instances the mode of onset is not specifically stated. I saw the greater number of these patients within the last five years and it is quite a frequent occurrence now for a patient to be brought to me and to be found to have a paralysed limb or muscle without any other history of onset of poliomyelitis. The frequency of scoliosis associated with paralysis of doubtful origin, probably due to poliomyelitis, must also be borne in mind. It is possible that the parents did not notice the acute symptoms, but at any rate these patients can be classified as coming under the nomenclature of "morning paralysis."

Yours, etc.,

N. D. ROYLE.

185, Macquarie Street,
Sydney,
March 19, 1931.

A WARNING.

SIR: On two occasions lately I have been rung up by doctors from North Sydney to tell me that a lad of about eighteen years of age has called upon them, stating that he is my son and is stranded without money. I have no son of that age, and the case is one of imposture. I have notified the North Sydney police, and if the young man should call on any one happening to read this, it would be wise for them to detain him and communicate with the police. He is described as being dark and with dark eyes. I have nothing but the vaguest suspicions as to who he is, but he is evidently someone who knows something of my movements of late years.

Yours, etc.,

SYDNEY J. WOOLNOUGH.

57, Macquarie Street,
Parramatta,
March 29, 1931.

Proceedings of the Australian Medical Boards.

VICTORIA.

THE undermentioned have been registered under the provisions of Part I of the *Medical Act*, 1928, of Victoria, as duly qualified medical practitioners:

Baylis, Edna Isabel, M.B., B.S., 1929 (Univ. Sydney), 40, Springdale Road, Killara, New South Wales.
 Carew-Smyth, Raymond Ponsonby, L.R.C.P. et S. (Edinburgh), L.R.F.P.S. (Glasgow), 1930, 180, Toorak Road West, South Yarra, S.E.1.
 Bryan, Claude Vivian Joseph, M.B., B.S., 1924 (Univ. Melbourne), 81, Garden Vale Road, Elsternwick, S.4.

Name restored to the Register:

Johnston, Leonard Walter, M.B. et Ch.B., 1918 (Univ. Melbourne), 22, Studley Avenue, Kew.

Books Received.

COLLECTED PAPERS OF THE BAKER INSTITUTE OF MEDICAL RESEARCH, ALFRED HOSPITAL, MELBOURNE: Number 1; 1930. Crown 4to.

Diary for the Month.

APR. 14.—New South Wales Branch, B.M.A.: Ethics Committee.
 APR. 21.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
 APR. 22.—Victorian Branch, B.M.A.: Council.
 APR. 24.—Queensland Branch, B.M.A.: Council.
 APR. 28.—New South Wales Branch, B.M.A.: Medical Politics Committee.
 APR. 30.—South Australian Branch, B.M.A.: Branch.
 APR. 30.—New South Wales Branch, B.M.A.: Branch.

Medical Appointments.

Dr. R. M. Gillies and Professor P. MacCallum (B.M.A.) have been appointed members of the Dental Board of Victoria for a period of three years pursuant to the provisions of the *Medical Act*, 1928.

Dr. F. Gallasch (B.M.A.) has been appointed Medical Officer of Health to the Kellerberrin Road Board, Western Australia.

Dr. G. R. Osborn (B.M.A.) has been appointed Government Medical Officer, Department of Mines, Western Australia, in accordance with *The Miners' Phthisis Act*, 1922.

Dr. F. W. Cotton (B.M.A.) has been appointed District Medical Officer and Public Vaccinator at Roebourne, Western Australia.

Dr. M. F. Williams (B.M.A.) has been appointed Medical Officer of Health to the Manjimup Road Board, Western Australia.

Dr. R. E. N. Byrnes (B.M.A.) has been appointed Public Vaccinator at Birregurra, Victoria.

Dr. A. D. Smith has been appointed Resident Medical Officer at the Government Hospital, Kalgoorlie, Western Australia.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc. see "Advertiser," page xvi.

CHILDREN'S HOSPITAL (INCORPORATED), PERTH, WESTERN AUSTRALIA: Junior Resident Medical Officer.

Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCH.	APPOINTMENTS.
NEW SOUTH WALES:	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Friendly Society Lodges at Casino. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane.	Members desiring to accept appointment in ANY COUNTRY HOSPITAL are advised to submit a copy of their agreement to the Council before signing, in their own interests. Brisbane Associated Friendly Societies' Medical Institute. Mount Isa Hospital. Mount Isa Mines.
SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide.	All Lodge Appointments in South Australia. All Contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.
NEW ZEALAND (Wellington Division): Honorary Secretary, Wellington.	Friendly Society Lodges, Wellington, New Zealand.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to "The Editor," THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

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